Osteoporosis: It's More Than Calcium

Jeanne Freeman[1] and Lori Turner[2]

[1]*California State University, Chico*
[2]*University of Arkansas*

Abstract

Osteoporosis is a major public health concern with millions of Americans being affected. This painful and oftentimes crippling disease is multifactorial in nature. Therefore, the development of osteoporosis is related to more issues than a person's intake of calcium. As a result, osteoporosis is not age or gender dependent. Risk factors for osteoporosis include proper nutrition including calcium intake, heredity, age, gender, physical inactivity, smoking, excessive alcohol use, and the use of several medications. With such a variety of risk factors, everyone should assume risk for disease development and thus take steps for the prevention of this bone fragility disease.

Keywords: osteoporosis, women's health, health education, calcium intake

Osteoporosis is a major public health concern in the United States. An estimated 28 million Americans are already afflicted with this disease and approximately 1.5 million new fractures occur each year (Barefield, 1996; Boughton, 1999; Krall & Dawson-Hughes, 1999; National Institutes of Health (NIH), 2000). In addition to those already afflicted, another 18 million Americans have low bone density, placing them at risk for this disorder (NIH, 2000).

Osteoporosis is a painful, sometimes crippling disease. The incidence of osteoporosis has reached epidemic levels in the United States and is responsible for considerable death, illness, loss of independence, decreased quality of life, and associated economic costs (Gerrior, Putnam, & Bente, 1998; National Osteoporosis Foundation [NOF], 1998). Estimates for the United States indicate that 13-18% of women over the age of 50 are afflicted with osteoporosis. Even more startling is the estimate that another 37-50% of women over 50 have some degree of osteopenia or low bone density (Barefield, 1996; Gerrior, et al., 1998).

Fractures that are a result of bone fragility account for extensive morbidity, mortality, and loss of function (Lappe, 1994; Turner, Taylor, & Hunt, 1998; Ullom-Minnich, 1999). The financial costs associated with osteoporotic fractures include direct medical charges, rehabilitation, and extended treatment facilities. Osteoporosis-related hip fractures alone result in estimated costs of $12.8 billion to $17.8 billion per year. Rehabilitation and institutionalization account for approximately 40% of these costs while less than 1 percent is due to lost productivity (Barefield, 1996). Rehabilitation and institutionalization costs are the largest majority since almost half of the individuals hospitalized with osteoporosis-related hip fractures never fully recover. Twenty-five percent of the total hip fractures result in the person being severely handicapped. Another 20% of these people die within one year of a hip fracture (Barefield, 1996; Lappe, 1994). As the population subgroup of Americans over the age of 65 grows, estimates indicate osteoporosis-related costs will be greater than $62 billion by 2020 and $200 billion by the year 2040 (Barefield, 1996; Cummings, Rubin, & Black, 1990).

Osteoporosis is a complex multifactorial condition characterized by low bone mass and

deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture (Notelovitz, 1993; Siddiqui, Shetty, & Duthie, 1999). Osteoporosis is often referred to as the silent thief because osteoporotic bone loss takes place gradually throughout the course of many years without any signs or symptoms (Boughton, 1999). This reduction in bone mineral density (BMD) later manifests itself as low-trauma fractures.

To better understand osteoporosis, it is helpful to understand the process of bone formation. Bone is living tissue that is constantly being renewed in a two-stage process of formation and resorption that occurs throughout life (Boughton, 1999). During different life stages, the balance of these processes may weigh heavier in one direction than the other. For example, during adolescence, bone formation occurs at a greater rate than bone resorption. The formation stage is characterized by the building of new bone to replace old bone by cells called osteoblasts. The resorption phase is characterized by a higher level of bone breakdown than formation as a result of cells called osteoclasts (Boughton, 1999; Smith, 1993).

There are two main type of bone that are recognized: trabecular or spongy bone (25%) and cortical or compact bone (75%) (Smith, 1993). Trabecular bone forms the internal support system of the bone and is metabolically more active than cortical bone (by about ten times) (Notelovitz, 1993; Smith, 1993; Wardlaw, 1993). Cortical bone makes up the outer shell of bone and predominates in the shafts of long bones. Although each bone in the body contains both cortical and trabecular bone, the relative proportions differ. In osteoporosis, both cortical thinning and a loss of trabecular support are evident (Wardlaw, 1993). However, trabecular bone loss occurs at a greater rate thereby increasing fracture risk in areas where trabecular bone makes up the largest portion of the bone's structure such as in the vertebrae, wrist, and the ends of long bones (i.e. the hip) (Boughton, 1999; Physicians Desk Reference (PDR), 1999; Smith, 1993; Wardlaw, 1993).

Osteoporosis is not always a result of bone loss and can be characterized as either primary or secondary in nature. Primary osteoporosis can occur in both genders at all ages but often follows menopause in women and occurs late in life in men (NIH, 2000). Postmenopausal osteoporosis is known as primary osteoporosis Type I and is characterized by an increased bone resorption that primarily affects trabecular bone. Type I primary osteoporosis is directly linked to the decreased production of estrogen that coincides with menopause (Peterson, 2001; Wardlaw, 1993). Rapid bone loss is osteoclast-mediated and occurs in women within the first 5 to 10 years after menopause (Peterson, 2001). Primary osteoporosis Type II is a slow bone loss resulting from a proportionate loss of trabecular and cortical bone usually due to a decrease in bone cell activity accompanying aging. This type of osteoporosis predominately afflicts men and women over the age of 70 years and is called senile osteoporosis (Glaser & Kaplan, 1997; Peterson, 2001; Wardlaw, 1993).

Secondary osteoporosis usually occurs as a result of another disease or medication. The most common medical conditions include chronic renal disease, hypogonadism, hyperthyroidism, Cushing's disease, and some forms of cancer (Wardlaw, 1993). Surgical procedures such as an early oophorectomy or total gastrectomy, can lead to bone loss. Additionally, some medications including anticonvulsants, corticosteroids, Depo-Provera, and Heparin, have toxic effects on bone and increase bone loss (Glaser & Kaplan, 1997; Kulak, Schussheim, McMahon, Kurland, Silverber, Siris, et al.., 2000; Peterson, 2001). Regardless of the cause of osteoporosis, the consequences are devastating to the financial, physical, and psychosocial aspects of one's health (NIH, 2000; Donohue, 1999; National Osteoporosis Foundation [NOF], 2000; Moon, 2000).

Researchers agree that osteoporosis is not age- or gender-dependent in who it targets. While white postmenopausal women have the highest incidence of osteoporotic fractures, and most of our knowledge about diagnosis and treatment is derived from research on this population, the

disease does not go unrecognized in other ages and races, or in men (Moon, 2000). However, in comparison to men, women are at higher risk for developing osteoporosis and have a lifetime risk of an osteoporotic fracture as high as one in three (Mark & Link, 1999). One third of women aged 65 and older will have at least one vertebral fracture in their lifetime and 33% will experience a hip fracture by age 90 (Aufdemorte, 1991). However, due to the diseases' silent nature, there are few reliable statistics on how many women, including young women, are already developing low bone density and osteoporosis. A study conducted by Tokar and colleagues among a convenient sample of 165 college-aged women found 1% of the participants to already have osteoporosis and an additional 14% of the participants to have low bone density or osteopenia (Tokar, Ford, Turner, & Denny, 2003).

Osteoporosis is a condition normally associated with elderly women. However, an increase in the number of women in their 20s and 30s suffering from osteoporosis has been reported (Hart & Dip, 1996). Among postmenopausal women, factors associated with osteoporosis diagnoses include age, race, and family history (Turner et al., 1998). Additional research indicates that other factors may also play a role in the development of this disease. Among these are factors that are common among women who are younger than the age of menopause. These other factors include low calcium intake, physical inactivity, smoking, excessive alcohol intake, use of steroid medications, and eating disorders (Hsieh, Novielli, Diamond, & Cheruva, 2001; Moon, 2000). The development of osteoporosis is associated with many risk factors that transcend age.

The prevention of osteoporosis is linked to strong bones being built during childhood and adolescence and being maintained throughout adult life (Mark & Link, 1999). Since clinical manifestations of osteoporosis often do not appear until later in life, one of the most important factors in preventing osteoporosis is the attainment of an optimal peak bone mass during adolescence and young adulthood (Cromer & Harel, 2000). Peak bone mass is defined as the highest level of bone mass achieved through normal skeletal growth (Masi & Bilezikian, 1997). The achievement of peak bone mass occurs in the third decade of life; about the age of 30 (Masi & Bilezikian, 1997; NIH, 2000; PDR, 1999). During the next 10-15 years, the bone structure stays relatively stable with slight reductions in mass if certain lifestyles are practiced. However, at the age of menopause, dramatic decreases in bone mass are lost due to changes in hormone production. Regardless of these hormonal changes, debilitating bone loss in not inevitable. The physiologic processes that lead to osteoporosis occur over much of a patient's lifespan and are amenable to interventions throughout that lifetime (Katz, & Sherman & DiNubile, 1998).

The optimization of bone health is a process that must occur throughout the lifespan because once a woman experiences a fracture due to bone fragility, no known therapy can rebuild the damaged bone to a healthy level. Therefore, measures taken to prevent bone fragility are of vital importance (Anderson & Metz, 1993; Blalock, et al., 1996). Evidence indicates that young women can increase their peak bone mineral density, promote long-term bone health, and reduce the risk of disease later in life by following effective dietary exercise and lifestyle practices (Mark & Link, 1999). Because there is currently no medical intervention to completely reverse the effects of osteoporosis, the most powerful tool to reduce the incidence of osteoporosis is prevention through health education (Mark & Link, 1999).

Osteoporosis prevention programs have traditionally been marketed toward women later in life (postmenopause). As a result, programs have emphasized nutritional changes, exercise programs, and hormone replacement therapy to prevent further bone loss. Few, if any, programs have been developed specifically for younger women as a means of preventing this debilitating disease (Blalock et al., 1996; Jamal, Ridout, Chase, Fielding, Rubin, & Hawker, 1999). This may be due to the seeming contradiction of young women having osteoporosis. However, if young women are to prevent or delay the development and onset of osteoporosis in later

life, then osteoporosis prevention needs to begin decades before women experience menopause (Kasper, Peterson, Allegrante, Galsworthy, & Gutin, 1994). A major component of such a prevention effort is education about behaviors that impact skeletal growth, the importance of regular menstrual cycles, proper nutrition, adequate physical activity, and cautions about medication use, smoking, and excessive alcohol intake.

Diet and Osteoporosis

Nutritional intake is a key component of osteoporosis prevention. Several different dietary factors play a role in either the advancement or prevention of osteoporosis while still others negatively effect bone mass.

Dietary Factors that Positively Effect Bone Health

Calcium. The roles of calcium in nature are numerous. This is also true when reviewing its roles in the human body. Calcium's most notorious role is that of structure or mechanics and is expressed in the mass, hardness, and strength of the bones and teeth (Weaver & Heaney, 1999). This is further evident with more than 99% of the calcium in the body being used and present in bones and teeth (Wardlaw, 1997). Overall, calcium accounts for 1-2% of a person's body weight (Weaver & Heaney, 1999).

The most documented and accepted health benefit of calcium is its role in bone health. In bone, calcium exists primarily in the form of hydroxyapatite ($Ca_{10}(PO_1)_6(OH)_2$) (Weaver & Heaney, 1999). When bones form, the calcium salts form crystals called hydroxyapatite, on a matrix of the protein collagen (Whitney & Rolfes, 2002). As the crystal structure becomes denser, the strength and rigidity of the bones increase.

Bone is a dynamic tissue that is constantly undergoing osteoclastic bone resorption and osteoblastic bone formation (National Academy Press (NAP), 2000). In growing children, bone formation exceeds resorption. This process is balanced in healthy adults while formation lags behind resorption after menopause and with aging in men and women (NAP, 2000). The skeleton has an obvious structural role and also serves as a reservoir for calcium.

Deficiency is the most widely known issue associated with calcium intake. A chronic inadequate calcium intake through diet or supplementation is one factor in the etiology of several disorders. The disorder given the most attention is osteoporosis. This disease is multifaceted with many correlated risk factors such as smoking, glucocorticoid use, and physical inactivity (Swaminathan, 1999). However, many of these risk factors influence calcium uptake and utilization. Yet the role of calcium intake in the prevention of osteoporosis can be reduced to two basic principles: build the highest peak bone mass possible and protect the bone mass that has accumulated (Heaney, 1992).

Low calcium intakes coupled with high obligatory calcium losses from the body, deplete calcium reserves. In other words, low intakes cause subnormal bone mass and strength. This is one of the contributing factors of osteoporosis. The primary strategies for reducing osteoporosis risk are to optimize bone mass during growth and to reduce bone loss later in life. The aim of both of these strategies is to maximize calcium intakes (Weaver & Heaney, 1999; Krall & Dawson-Hughes, 1999). In the past few years, evidence has been given to indicate that dietary calcium intake is positively related to bone mineral density in children and adolescents. Research indicates the higher the calcium intake, the greater the peak bone mass (Valimaki et al., 1994; Jackman et al., 1997). Additionally, research has indicated a positive correlation between bone mass and calcium intake in premenopausal adult women (Welten, Kemper, Post, & Van Staveren, 1995). Overall, there is strong evidence that calcium intake influences bone mass in all age groups (Gennari, 2001).

Adequate dietary calcium is essential for building denser, stronger bones in the first three decades of life and for slowing the rates of bone loss in later years (Gerrior et al., 1998). The importance of calcium in the human diet is evident in many government publications. Healthy People 2010 is a document created to

identify the current health status of Americans and the health and lifestyle areas needing improvement. One of the goals of the Healthy People 2010 document is to increase the quality of life among Americans. This is important when evaluating calcium intake and its role in the development of osteoporosis. As a result, there are three calcium-related objectives outlined in Healthy People 2010. One of these is specific to calcium intake while the other two are related to osteoporosis.

Objective 19-11. Increase the proportion of persons aged two years and older who meet dietary recommendations for calcium.

Calcium is essential for various mechanic and physiologic functions in the body. According to the Institute of Medicine (IOM) (1997), and the new Dietary Reference Intakes, the recommendations for adequate daily intakes (AIs) of calcium are 500 milligrams for children one to three years. The recommendations for other age groups include children aged four to eight years, 800 milligrams; for adolescents aged nine to 18 years, 1300 milligrams; for adults 19 to 50 years, 1000 milligrams; and for adults older than 51 years, 1200 milligrams (IOM, 1997).

According to baseline data collected during the National Health and Nutrition Examination Survey of 1988-1994 (NHANES III), only 46% of persons aged two years and older were at or above approximated mean calcium requirements based on calcium from foods, dietary supplements, and antacids. By the year 2010, the target for adequate calcium intake is 75% of the population agedtwo years and older.

Sources of dietary calcium are numerous. The most often recognized form is that of dairy products. Milk products contribute a significant proportion of calcium to the diets of women in western societies and women who do not consume milk products are unlikely to meet their calcium needs (Fleming & Heimbach, 1994; Horwath, Bovern, Campbell, Busby, & Scott, 1995). Several barriers to milk product consumption have been identified in research. Low consumption has been associated with a dislike for milk products (Horwath et al., 1995), adverse reactions such as lactose intolerance (Arney & Pinnock, 1993), perceived adequate intake (Chapman, Chan, & Clark, 1995), and the perception of milk products increasing dietary fat and cholesterol intake (Horwath et al., 1995). However, research has also identified that consuming more meals at home or at sit-down restaurants is associated with moderate calcium intake (Lewis & Hollingworth, 1992).

The barrier to dairy product intake related to increasing fat and cholesterol is important to highlight. Many young women engage in self-imposed energy reduction diets. As a result, they are at risk for not obtaining an adequate calcium intake level. In a study conducted in Australia, researchers found that 68% of the 18-year-old university students in the study had calcium consumption levels below 800mg. The majority of these women were on energy-reduction diets and at risk of a dietary calcium intake deficit at a time when calcium intake should be enhanced (Portsmouth, Henderson, Graham, Price, Cole, & Allen, 1994). Dietary modification in the form of dairy products is important to consider. A three-year prospective study found that increasing dairy product consumption retards vertebral bone loss in premenopausal women (Baran et al., 1989). Dairy products are the richest source of dietary calcium and need to be consumed to assist women in achieving adequate calcium intake. In fact, research indicates the most people obtain 50% of their calcium from dairy products (Wardlaw & Weese, 1995).

Calcium supplementation is another effort utilized to meet the published Healthy People 2010 objectives. Interest and enthusiasm in the use of dietary supplements appears to be growing in the United States. According to NHANES III data collected in 1988-1994, approximately 40% of the population took dietary supplements during the month prior to the interview (Ervin, Wright, & Kennedy-Stephenson, 1999). Characteristics of these supplement users included women, Caucasian, increasing age, higher levels of income and education, and a self-reported health status of good or fair. Of these supplement users,

approximately 46 percent took a combination vitamin/mineral product (Ervin et al., 1999). The data did not indicate the proportion of people consuming calcium supplements alone.

Calcium supplements are a nondietary, alternative source of calcium (Sutton, 2000). Individuals who are unable to get enough calcium in their regular diet generally take calcium supplements. These nondietary alternatives often come as a salt. A calcium "salt" contains calcium along with another substance, such as carbonate or gluconate (Micromedex, 2001). As a result, some calcium salts have more elemental calcium than others.

Studies examining the effects of calcium supplementation in premenopausal women have shown conflicting results. Some have shown no significant effect on bone mineral density (BMD) in premenopausal women (Smith, Gilligan, Smith, Surpos, 1989) while still others have shown significant increases in BMD (Baran et al., 1989; Rico, Revilla, Villa, de Buergo, & Arribas, 1994; Lloyd et al., 1993; Johnston et al., 1992). Although there have been discrepancies in the findings, the overall conclusion is that calcium intake, even from supplements, has some effect in increasing peak bone mass during growth and bone maturation.

Although calcium supplements have a role in achieving adequate calcium intake, it is important to remember that the best source of calcium is food. A calcium supplement is to supplement what is obtained from food sources. Additionally, it is important to remember that bone health is not based on a single nutrient (Heaney, 1992). Diets low in calcium also tend to be low in other nutrients that are essential for normal bone development such as zinc, manganese, copper, ascorbic acid, protein, and vitamin D (Holbrook, & Barrett-Connor, 1991).

The need for knowledge is extensive. Calcium intake is vital for the current and future health of individuals. Information about how to obtain adequate calcium intakes is vital for several subpopulations such as vegetarians, lactose intolerant individuals, ethnic groups, and young women. Education about calcium needs to be expanded throughout one's life because research indicates that bone mass can increase through the third decade of life (Recker, Davies, Hinders, Heaney, Stegman, & Kimmel, 1992).

Vitamin D. Vitamin D plays an important role in calcium metabolism, calcium absorption, and bone health. The National Osteoporosis Foundation describes the relationship between calcium absorption and vitamin D as being similar to that of a locked door and key (NOF, n.d.). Vitamin D is the key that unlocks the door and allows calcium to leave the intestine and enter the bloodstream. Vitamin D also works in the kidneys to help resorb calcium that otherwise would be excreted (NOF, n.d.).

Vitamin D is a fat soluble vitamin that has been associated with bone-related disorders for centuries. Vitamin D deficiency is associated with rickets in children, osteomalacia in adults, and secondary hyperparathyroidism (Combs, 1998). The daily requirement for this vitamin is met from the diet or from synthesis in the skin. From either of these sources, vitamin D is metabolized to 25-hydroxy vitamin D (25OHD) in the liver and is then converted to the active metabolite 1,25 dihydroxyvitamin D (1,25 [OH2 D) in the kidney (Combs, 1998; Swaminathan, 1999).

Recommendations for vitamin D intake are 200 international units (IU) for women under the age of 50, 400 IU for women 51-70 years of age, and 600 IU for women over 70 years of age (Willhite, 1998). The increase in vitamin D intake recommendations indicates that ageing affects vitamin D metabolism. The conversion of 25OHD to the active metabolite 1,25(OH)2D is reduced because of an age-related decline in renal function (Swaminathan, 1999). Additionally, as adults age, the ability to make vitamin D through the skin decreases (NOF, n.d.).

There are many sources of vitamin D available, however sunshine is the major source. One study indicated that total body sun exposure could provide the equivalent of 10,000 IU of vitamin D without a person experiencing toxicity. The study suggested that the RDA for

vitamin D is a physiologic limit (Vieth, 1999). It may be important to note that sources of vitamin D may be managed differently in the body and therefore, the body can handle a greater amount of sunlight produced vitamin D than vitamin D from dietary sources. However, dietary sources of vitamin D are necessary to utilize by people who do not get adequate sun exposure due to latitude, season, work environment, etc. Dietary sources of vitamin D include vitamin D-fortified dairy products, egg yolks, saltwater fish, liver, and supplements (Dowd, 2001; NOF, n.d.; Willhite, 1998).

Vitamin D enhances calcium's ability to build and maintain bones. Several studies have indicated the importance of vitamin D as a partner with calcium in the prevention of osteoporotic fractures. Vitamin D in combination with calcium has been shown to increase bone density and decrease fracture rates (Dawson-Hughes, Harris, Krall, & Dallal, 1997; Chapuy et al., 1992). Additionally, vitamin D supplementation has demonstrated an increase in bone mineral density (Dawson-Hughes, Harris, Krall, Dallal, Falconer, & Green, 1995; Ooms, Roos, Bezemer, Van Der Vijgh, Bouter, & Lips, 1995).

Vitamin D has a vital role in the health of bone. The role of this fat soluble vitamin is important for the absorption and utilization of calcium which is the primary mineral in bone. Therefore, it is important for individuals to be knowledgeable about the interaction of this vitamin and mineral for total bone health. Young women run the risk of being deficient in vitamin D if they avoid the sun or fail to consume adequate dietary sources. In other words, if women avoid consuming dairy products, they not only lose out on the calcium content but also the possibility for vitamin D consumption.

Fluoride. Fluoride has effects on the matrix of bone itself and on osteoblast function. Although fluoride has been used for dental carry prevention, it is approved for the treatment of osteoporosis in many countries around the world (Dowd, 2001; Sowers, Wallace, & Lemke, 1986). Fluoride is capable of stimulating osteoblasts to increase bone formation. Although the new bone has increased crystallinity, making it more resistant to resorption, the bone formed is abnormal and mechanical strength is compromised (Willhite, 1998). A clinical trial study demonstrated this compromise of mechanical strength by utilizing immediate-release fluoride. The study showed gains in spinal bone mineral density but spinal fracture rate was not decreased. Additionally, fracture risk of the hip and appendicular skeleton actually increased (Pak, Sakhaee, Rubin, Zerwekh, 1997). The authors concluded that fluoride may increase trabecular bone density at the expense of cortical bone even with adequate calcium supplementation (Pak et al., 1997).

Additional studies have been conducted using sustained-release fluoride. A study conducted by Pak and colleagues (1995) looked at osteoporotic women consuming sustained-release sodium fluoride and calcium citrate for four years. The study found an increase in spinal BMD by almost 5% and a decrease in vertebral fracture rate when compared to calcium intake alone (Pak, Sakhaee, Adams-Huet, Piziak, Peterson, Poindexter, 1995).

Fluoride is a compound that aids in the development of a strong bone structure. However, studies indicate that fluoride alone can actually reduce bone strength. The research further indicates that in combination with other minerals and vitamins such as calcium and vitamin D, fluoride has its greatest benefits for bone health.

Other positive dietary factors. Several other vitamins and minerals play a key role in the health of bone and therefore the prevention of osteoporosis. It is important to discuss these compounds because it supports the fact that bone formation and health is a complex and multifaceted process. It is important for one to have good overall health and to achieve such a status through adequate dietary intake. Simply taking supplements of various vitamins and minerals does not allow for adequate or appropriate nutritional balance to assist in adequate bone health.

Vitamin K has been suggested to play a specific role in osteoporosis. Vitamin K is involved in the synthesis of various proteins in the body including three in bone tissue: osteocalcin, matrix gla protein (MGP) and protein S (Dowd, 2001; Swaminathan, 1999). Vitamin K-dependent proteins contain gamma carboxyglutamic acid residues, and vitamin K is required for the carboxylation reaction of glutamic acid (Combs, 1998; Swaminathan, 1999). A lack of vitamin K will lead to a reduction in carboxylation of vitamin K-dependent proteins. The concentration of undercarboxylated osteocalcin (UcOC) has been reported to be higher in postmenopausal women than in premenopausal women (Knapen, Hamulyak, & Vermeer, 1989) and there seems to be an increase in UcOC with age (Plantalech, Guilaumont, Vergnaud, Leclercq, & Delmas, 1991). Additionally, elevated UcOC concentrations have been associated with low bone mass in the femur and an increased risk of hip fracture in elderly women (Szulc, Arlot, Chapuy, Duboeuf, Meunier, & Delmas, 1994; Szulc, Chapuy, Meunier, Delmas, 1993).

Vitamin K is also known for its role in blood clotting. For those who are taking anticoagulants, caution needs to be exercised when taking supplemental Vitamin K because it could affect clotting. A physician should be consulted prior to taking supplemental vitamin K. The major dietary source of vitamin K is green vegetables (Combs, 1998). Although vitamin K deficiency is rare in most societies and its role in bone health is not completely clear, vitamin K cannot be ignored as a compound of importance in bone health.

Phosphorus is another compound that is important for bone health. Phosphorus is widely available in the diet and is part of the crystal structure of bones. Almost 85% of the body's phosphorus is present in crystalline form in bone as hydroxyapatite (Gennari, 2001). Since phosphorus is so abundant in the diet, a nutritional deficit is generally not a concern. However, among those with malnutrition or low dietary calcium intakes, phosphorus may bring harm by causing an increased calcium excretion (Dowd, 2001).

Phosphorus deficiency may be more important than currently recognized. Multiple nutritional deficiencies have been implicated in the concentrated number of fractures of the upper femur (Combs, 1998). With approximately 20% of elderly individuals in industrialized nations ingesting less than 60% of the RDA for phosphorus or protein, a relative hypophosphatemia might result (Heaney, 2000). This inadequate phosphorus intake could slow bone repair of osteoporotic fractures (Dowd, 2001). When searching for a good source of phosphorus, it is important to know that milk provides calcium and phosphorus in an optimal ratio for building bone tissue (Dowd, 2001).

The final mineral to be discussed in relation to bone health is magnesium. Approximately 50-60% of the magnesium in the body is in bone (Gennari, 2001). A well-balanced diet including whole grains, legumes, green leafy vegetables, and nuts contains a healthy amount of magnesium (Dowd, 2001). Although many people do not consume an adequate amount of calcium in their diet, magnesium deficiency is relatively rare (Combs, 1998; Dowd, 2001).

In experimental magnesium deficiency studies, osteoblastic and osteoclastic activity is decreased and there is a cessation of bone growth and osteopenia develops (Robbins & New, 1997). Additionally, the magnesium content of trabecular bone in osteoporotic subjects is significantly lower than other subjects and magnesium intake has been reported to be lower in osteoporotic subjects (Combs, 1998; Schwartz & Reddi, 1979).

In addition to its content in bone, magnesium plays a role in the formation of 1,25 dihydroxyvitamin D. A magnesium-dependent hydroxylase enzyme is involved in the formation of 1,25 dihydroxyvitamin D. Therefore, if a magnesium deficiency did occur as in those with serious illness, alcoholism, prolonged vomiting and diarrhea, or intestinal malabsorption, this could possibly adversely affect calcium absorption and therefore bone strength (Combs, 1998; Dowd, 2001; Risco, Traba, de la Piedra, 1995).

Dietary Factors that Negatively Effect Bone Health

Protein. Protein-energy malnutrition during childhood or adolescence may retard growth and reduce body strength and peak bone mass (Toss, 1992). However, it has also been suggested that a high-protein diet may increase the risk of osteoporosis as a result of increases in urinary calcium excretion (Lau & Woo, 1998). The mechanism of increased urinary calcium excretion is thought to be a result of glomerular filtration rate (GFR) and the acid load from the sulphur containing amino acids methionine and cysteine (Marcus, 1982). This increase in endogenous acid production mobilizes calcium from the skeleton to form salts to neutralize the acidity (Krieger, Sessler, & Bushinsky, 1992).
Concerns about high-protein diets arose when studies showed increases in urinary calcium excretion (Heaney, 1993). Although there is little evidence from clinical trials to support the observation that high dietary protein intake causes increased bone loss, result of observational epidemiologic studies do support this view. Results from two ecologic studies indicate that the incidence of hip fracture is inversely related to the per capita consumption of protein (Abelow, Holford, & Insogna, 1992; Hegsted, 1986). While it is difficult to interpret these results because of the numerable confounding effects of other dietary components, these studies still lend concern to the issue of protein intake and osteoporosis development.

The average protein intake in many industrialized countries, including the U.S., is at least 50% above recommended levels (Krall & Dawson-Hughes, 1999). An average increase in dietary protein of 1 gram results in the loss of an additional 1 milligram of calcium in the urine (NAP, 2000; Krall & Dawson-Hughes, 1999). Such calcium losses could be important for individuals with a low usual calcium intake or impaired calcium absorption. Current recommendations would result in a dietary calcium/protein intake ratio of 20mg calcium/1 g protein (Heaney, 1998; IOM, 1997).

While protein has been shown to increase calcium excretion, its roles in calcium absorption and retention are controversial. A twenty-year study conducted by Heaney (2000), on Catholic nuns, concluded that protein intakes, within the current U.S. intake levels, do not affect calcium absorption. The research related to protein's influence on osteoporosis is still controversial, but two things are known for sure: a) protein is necessary for bone and muscle health, and b) high levels of protein intake increase urinary calcium excretion (Powers et al., 1999).

Sodium. Sodium and calcium excretion are linked in the proximal renal tubule. Sodium causes an increase in renal calcium excretion (Krall & Dawson-Hughes, 1999). For each 500-milligram increment in sodium excretion or intake, there is approximately a 10-milligram increase in the amount of calcium lost in urine (NAP, 2000).

A study conducted by Devine and colleagues (1995) on postmenopausal women, demonstrated the impact of sodium intake on the rate of bone loss at the hip. The study identified an increasingly negative change in hip bone density with higher urinary sodium levels and with increasing sodium intakes between 1 and 6 grams per day. The study further suggested that halving the current sodium intake of 121 mmol/d (or 2700 mg/day) would be equivalent to increasing dietary calcium by 22 mmol/d (or 890 mg/day) (Devine, Criddle, Dick, Kerr, & Prince, 1995). However, a study of pubertal females found no association between bone mineral density and urinary sodium excretion (Matkovic et al., 1995).

Studies correlating high sodium intake with a decrease in bone mineral density raise concern due to the high salt content of processed foods and the quantity of these foods that Americans consume. Reassuring information from a study by Massey and Whiting (1996) was that calcium intakes between 1000 milligrams and 2000 milligrams minimized bone loss in the hip area associated with diets high in sodium. These calcium levels are within the new Dietary Reference Intakes (DRIs) recommendations and do not exceed the Tolerable Upper Limit values (ULs).

Caffeine. The consumption of caffeine and its relationship to bone health is a controversial topic. Caffeine is the most widely consumed psychoactive substance in the world with coffee supplying greater than 80% of the caffeine consumed by adults in the United States (Barone & Grice, 1994). Numerous studies have reported on caffeine as a possible risk factor for bone loss in adult women. The results however have been contradictory.

Several studies have reported no association between caffeine intake and fracture frequency or changes in bone density (Johansson, Mellstrom, Lerner, & Osterber, 1992; Lloyd, Rollings, Eggli, Kieselhorst, & Chinchilli, 1997; McCullock, Bailey, Houston, & Dodd, 1990; Tavani, Negri, & LaVecchia, 1995). Others, however, have reported small but significant increases in either fracture frequency or bone loss. The most notable of these studies is the Framingham study that reported a 53% greater incidence of hip fracture in those who consumed more than two cups of coffee or four cups of tea after controlling for weight, sex, age, estrogen use, smoking and alcohol (Kiel, Felson, Hannan, Anderson, & Wilson, 1990). Another noteworthy study was the Nurses Health Study of 84,000 women who were followed for 6 years. The study found that women with the highest intake of coffee and the highest intake of caffeine (800 mg or more daily) had three times the rate of hip fractures as the no coffee/no caffeine group (Hernandez-Avila, Colditz, Stampfer, Rosner, Speizer, & Willett, 1991).

Caffeine ingestion causes a short-term (within 1—3 hours) increase in urinary calcium loss, but studies have failed to document sustained effects of caffeine on urinary or fecal calcium excretion (Krall & Dawson-Hughes, 1999). Among people who have low calcium intakes the effect may be of great importance as the body fails to adequately compensate for the additional calcium loss.

The effects of caffeine on bone mass in young women have also been a controversial topic. A study conducted by Packard and Recker (1996) indicated that a moderate caffeine intake (one cup of coffee per day or 103 mg) appeared to be a safe level with respect to bone health. However, another study conducted by Conlisk and Galuska (2000) did find that caffeine consumption decreased bone mineral density at various skeletal sites. The study indicated that for every 100 mg of caffeine consumed, femoral neck BMD decreased 0.0069 g/cm2, and lumbar spine decreased 0.0119 g/cm2 (Conlisk & Galuska, 2000). Although there was no significant difference between those who consumed low levels of calcium and those who consumed high levels of calcium in this study, it stands to reason that such decreases over many years can increase a woman's risk of osteoporosis significantly.

Although sustained effects of caffeine on calcium excretion have not been observed in these studies, there is another aspect of this consumption that needs consideration; beverage replacement. On any given day, half of all Americans drink carbonated soft drinks according to data collected for 1994-1996 (Gerrior et al., 1998). The intake of these beverages has increased drastically among teenagers, younger adults, and women drinking low-calorie beverages, since 1970. Annual food supply data show that per capita consumption of regular carbonated soft drinks increased from 22 gallons in 1970 to 40 gallons in 1994 and to 41 gallons in 1997 (Gerrior et al., 1998). Whether caffeine affects over all calcium excretion is important, but the fact that carbonated beverages are replacing calcium-rich drinks needs consideration.

Long-term effects of frequent caffeinated beverage intake must be conducted before a consensus about the true relationship between caffeine and calcium can be decided upon. If caffeine increases calcium excretion for 1-3 hours, it stands to reason that a person who consumes 3-4 caffeinated beverages a day will have a significant loss of calcium in their urine.

Dietary Factors with Questionable Effects on Bone Health

Soy and isoflavones. Phytoestrogens such as isoflavones, which are found in many soy foods and supplements, have a chemical structure that causes them to act in the body like the estrogenic

hormone estradiol (Anderson, 1999; Messina, 1995; Messina, 1999). The lower rate of hip fracture among Japanese women in comparison to US women is often cited as providing support for the protective effect of isoflavones (Ross et al., 1991). However, this argument is without merit (Messina, 1999).

There is some evidence that soy may reduce bone turnover as measured in urine and serum bone markers (Alekel, St. Germain, & Peterson, 2000; Pennington & Schoen, 1996; Messina, Gardner, & Barnes, 2002). However, researchers at Creighton University found no effect on spine and femur bone mineral density in early postmenopausal women (Heaney, Dowell, Rafferty, & Bierman, 2000). Although soy has nutritional value, its effects on bone seem to be small and of tentative clinical importance.

Several studies have been conducted to compare the antiresporptive effect of estrogen replacement in postmenopausal women versus that of soy isoflavones (Heaney et al., 2000; Setchell, 2000). However, in both of these studies, there was not a greater bioavailability of soy isoflavones. In fact, calcium in fortified soy milk was not absorbed as efficiently as calcium from non-soy milk.

Although several animal studies indicate a potential benefit in the inhibition of bone resorption as a result of soy isoflavones (Anderson, Ambrose, & Garner, 1995; Brandii, 1992) there have not been consistent results when applied in human models. There are several areas left unanswered that are necessary to delineate the effects of soy fully or to allow making any recommendations (Dowd, 2001). Information regarding efficacy, safety, which isoflavones have the greatest effect, and amounts of isoflavones necessary for measurable benefit, are necessary to provide accurate recommendations.

Conditions That Effect Dietary Intake

Lactose Intolerance. About 25% of adults in the United States have lactose intolerance and develop symptoms of diarrhea and bloating after ingestion of a large dose of lactose (NAP, 2000). This condition is caused by a deficiency of an enzyme that breaks down milk sugar in the intestine called lactase. Many people believe they are lactose intolerant, but they do not know they do not have to be. Lactose intolerance is especially common in African Americans, Hispanics, Native Americans, and Asian Americans (Gerrior et al., 1998). Those who demonstrate lactose intolerance often avoid dairy products entirely. However, such foods as hard cheese (Swiss, cheddar, American), yogurt, and lactose-reduced milk have lower lactose levels and therefore can be consumed by sufferers of lactose intolerance (Dowd, 2001). In addition, studies indicate that many lactose-intolerant people can tolerate smaller doses of lactose such as the amount present in an 8-ounce glass of milk without the negative side effects (Levin, 1999; NAP, 2000).

Lactose-intolerant individuals absorb calcium normally from milk, but they are at an increased risk of calcium deficiency because of their avoidance of milk and other calcium rich dairy products (NAP, 2000). Although lactose intolerance may influence intake, there is no evidence to suggest that it influences the calcium requirements (NAP, 2000). Therefore, to aid in calcium intake, lactose-free dairy products are available. There are also good nondairy sources of calcium such as canned salmon with the bones, fortified cereals, and calcium-fortified orange juice.

Nutritional Summary

Nutrition plays a significant role in the cause and prevention of osteoporosis. This disease can be prevented by maintaining an optimal calcium intake, maintaining an adequate vitamin D level, avoiding a high salt intake, and avoiding extremely high animal protein intakes. Nutrition through food consumption is the most adequate method to achieve good bone strength. The bone matrix is complex and simply taking a supplement or several supplements to possibly compensate for an inadequate diet, could cause a greater imbalance and still lead to a weak bone mass. Nutritional factors alone do not completely explain the prevention puzzle of osteoporosis. Additional factors such as physical activity, heredity, and ethnicity play a

role in understanding the etiology and prevention of the disease.

Lifestyle Factors and Osteoporosis

Physical Activity

Bone is living tissue that responds to exercise by becoming stronger and denser. There are two types of exercises that are important for building and maintaining bone strength and density: weight-bearing and resistance exercises (NOF, 2000). The specific characteristics of physical activity that are most important for influencing bone are not completely understood, but research indicates that high mechanical loads may be more osteotropic than low-intensity loads (Taaffe, Robinson, Snow, & Marcus, 1997). Additionally, the number of repetitions has been shown to make modest effects of bone mass (Carter, Fyrie, & Whalen, 1987; Rubin & Lanyon, 1985).

However, conclusions about the effects of exercise in the prevention of osteoporosis are still vague. Research seems to be inconclusive about the types of recommendations that should be made. The majority of osteoporosis researchers would probably agree that recommending physical activity is important for improving balance, muscle tone, flexibility, strength, and coordination; all aspects that could prevent falls and low-trauma fractures (American College of Sports Medicine [ACSM], 1995). Yet questions remain as to what types of activities, for how long, how often, at what intensity, and should the activities be site specific? Much research exists that indicates simply physical activity in itself can help prevent osteoporosis.

As with other areas of osteoporosis research, a lot of information is available for older populations. The few studies that exist for younger, premenopausal women lend support to the information that has been acquired while studying older populations. In a study conducted by Turner and colleagues, women who participated in the NHANES III study over the age of 50 were reviewed. The study identified physical activity as a greater predictor for fracture than heredity, smoking status, alcohol use, and dairy product intake (Turner, Leaver-Dunn, DiBrezzo, & Fort, 1998). The study revealed that inactive women were 84% more likely to suffer a fracture than females who were active 2 or more times per week.

Another study conducted by Mitchell and colleagues followed 2,567 women for an average of 11 years. This study reviewed the risk of developing osteoporosis based on cardiorespiratory fitness. The study revealed that the more fit a woman was, the less likely she was to develop physician diagnosed osteoporosis during the time frame of the study. After the researchers adjusted for age, body mass index, high blood pressure, cigarette smoking, alcohol consumption, and diabetes, they found that low fit women were 1.8 times more likely to develop osteoporosis than moderate or high fit women (Mitchell, Wei, Gibbons, & Blair, 1999).

Studies reviewing the types of activities women participate in and their risk for osteoporosis have also been conducted. One study recently published by Turner and colleagues (2002) indicated that yard work and weight training were strong and independent predictors for positive bone density. Again, data from the Third National Health and Nutrition Examination Survey (NHANES III) were used to study the relationship between exercise mode, frequency, and bone health (Turner, Bass, Ting, & Brown, 2002). Other activities were also identified as moderate predictors for positive bone density such as bicycling, aerobics, walking, and dancing (Turner et al., 2002).

Most of the data related to young adult women between the ages of 18-35 comes from cross-sectional studies comparing bone mineral density (BMD) of female athletes to that of a sedentary group. Direct measurements of bone mass have shown a positive correlation between spinal BMD and reported leisure time activity in healthy young women (Kanders, Dempster, & Lindsay, 1988). In this study, calcium and physical activity were independent determinants of BMD.

There have been few prospective studies of an exercise effect on bone mass in this age group.

In a study conducted by Snow-Harter and colleagues (1992), jogging or aerobic exercise for 20-30 minutes three times a week increased lumbar spinal bone mineral density by 1 percent in premenopausal women. Additionally, a study conducted by Bassey and Ramsdale (1994) found a significant increase of 3.4% in trochanteric bone density in a group of high-impact exercisers when compared to low-impact exercisers. Although these increases may seem to be small, any increase in bone mineral density that can delay the onset of osteoporosis or the complications of low bone density are important to consider and should not be discarded as being without merit.

Studies of athletes show that the BMD of loaded bones can be more than 30% higher in most studies and between 5% and 20% higher in most sites than that of unloaded bone or of the same bones in non-athletic control subjects (Vuori, 1996). However, for young women who are high-performance athletes, physical activity may increase their risk of osteoporosis. Intensive training that results in an extreme loss of body fat, disordered eating and estrogen deficiency can lead to bone loss (Vuori, 1996). The combination of osteoporosis, menstrual irregularities, and disordered eating is known as the female athlete triad (Sabatini, 2001).
Many young female athletes experience a cessation in their menstrual cycles called amenorrhea. Several studies have reported low bone densities among these women (De Cree, Vermeulen, & Ostyn, 1991; Licata, 1992; Fabbri et al., 1991). Low bone densities result in a greater risk for stress fractures and other more devastating fractures of the hip and spine (Bass, Turner, & Hunt, 2001). Furthermore, research has indicated that premature osteoporosis occurring in female athletes may be irreversible even with calcium supplementation, resumption of menses, or estrogen replacement therapy (Drinkwater, Bruemner, & Chestnut, 1990).

The extent of exercise's influence on osteoporosis and its ability to induce bone density increases is still uncertain. Results from these studies vary according to age, hormonal status, nutrition, and exercise prescription. Regardless of the uncertainty from research results, expert panels such as the American College of Sports Medicine, recommend weight-bearing activity and activities that improve strength, flexibility, and coordination to prevent osteoporosis and falls respectively (ACSM, 1995).

Alcohol Intake

Studies have indicated that alcohol suppresses bone formation. Since women are more prone to osteoporosis than men, the effects of alcohol may have a greater effect on their bones (Laitinen, Karkkainen, Lalla, Lamber-Allardt, Tunninen, Tahtela, et al., 1993). The evidence for the adverse effects of alcohol on bone mineral density (BMD) comes primarily from case control studies. However, in a 14-year longitudinal study, the rate of bone loss in men who drank regularly was faster than in controls (Slemenda, Christina, Reed, Reister, Williams, & Johnston, 1992). Similarly, BMD in premenopausal women who drank regularly was also found to be lower than in matched controls (Arden, 1997).

Alcohol is capable of increasing one's risk of osteoporosis or fracture through several different methods. First, alcohol abuse may bring about hypoestrogenism with consequent menstrual irregularities and amenorrhea (Mello, Mendelson, & Teoh, 1989; Van Thiel & Gavaler, 1990). These irregularities can depress osteoblastic activity (Lappe, 1994). This can lead to an imbalance between the resorptive osteoclastic activity and the formative osteoblastic activity progressing to decreased bone mineral density (Thomas, 1997).

Alcohol can also lead to interference with proper nutrition, especially calcium and vitamin D intake. Individuals who consume moderate to excessive amounts of alcohol often have an imbalanced diet with a decreased consumption of calcium (Wardlaw & Weese, 1995). Additionally, research has revealed that alcoholics have a reduced ability to produce 1,25 dihydroxyvitamin D in the renal tubules (Wardlaw & Weese, 1995). By consuming a calcium deficient diet and not having an adequate capacity to produce vitamin D, which is necessary for calcium absorption, the

consumption of alcohol can lead to an inability of the bone to reach its maximum strength.

A final way that alcohol can increase one's risk of fractures is due to balance difficulties experienced during inebriation which can increase the risk for falls. If a woman with poor bone health falls, she is more likely to break a bone than a women with good bone health (Thomas, 1997). All in all, alcohol may not have a direct influence on bone health, but through indirect means, a lifestyle that involves alcohol intakes of more than one drink per day can increase one's risk of osteoporosis or osteoporotic fracture (Laitinen & Välimäki, 1993).

Cigarette Smoking

Smoking puts women at risk for osteoporosis because smoking decreases serum estrogen (Thomas, 1997). A loss of estrogen leads to a decreased osteoblastic action progressing to an imbalance between resorption and formation. Estrogen also plays a role in the absorption of calcium, an essential nutrient in the formation of strong bones (Wardlaw & Weese, 1995). Additionally, smokers tend to have leaner body masses perhaps because of the interference of smoking with eating (Mazess & Barden, 1991). This combination of low estrogen and low body weight can lead to an increase in risk for osteoporosis.

Smoking is recognized as a risk factor for vertebral, forearm, and hip fractures (Hollenbach, Barrett-Connor, Edelstein, & Holbrook, 1993). Previous research indicates that women, who smoke throughout adulthood will, by the time of menopause, have an average deficit of 5 to 10 percent in bone density (Hopper & Seeman, 1994). Additionally this same study found that for every 10 pack-years of smoking, a 2% decrease in lumbar spine BMD, 0.9% decrease in femoral neck BMD, and 1.4% decrease in femoral shaft BMD were observed. Such percentages are sufficient to increase the risk of fracture.

As with other diseases associated with smoking, cessation can result in some positive effects. A study conducted by Cornuz and colleagues (1999) observed that smokers are at an increased risk of hip fracture and their risk rises with greater cigarette consumption. However, the risk declined among former smokers, but the greatest benefit was not observed until 10 years after cessation (Cornuz, Feskanich, Willett, & Colditz, 1999). The authors did identify that part of the benefit realized was associated with a difference in body weight.

The influence of both alcohol and smoking on bone health is complex. In reviewing studies regarding these lifestyle factors, it is important to realize the multiple potential confounding variables: age, heredity, consumption amounts, dietary intake, physical activity, etc. Although it is unlikely we will see a heavy smoker participating in the recommended levels of physical activity for general health, one cannot ignore the confounders present when reviewing the impact of smoking and alcohol intake on bone density.

Heredity and Osteoporosis

Osteoporosis is a multifaceted disease. One of the potential risk factors associated with disease development is family history. Lindsay and Dempster (1985) state that 75% of osteoporosis cases have a family history component. However, the degree to which this is due to genetics or to environment is debatable (Wardlaw, 1988). The most commonly cited studies supporting a genetic component are twin studies. These studies have estimated the heritability of bone density of the lumbar spine to be as high as 92% in premenopausal women (Pocock, Eisman, Hopper, Yeates, Sambrook, & Elberl, 1987) and around 70% for the femoral neck (Slemenda, Hui, Longcope, Wellman, Johnston, 1990). Additionally, other twin studies have shown a genetic effect but after the age of 25, the effect was no longer significant (Dequeker, Nijs, Verstraeten, Geusens, & Gevers, 1987). This might suggest that other factors such as the environment play a role after a certain age.

Research indicates that women who have a family history of osteoporosis generally have lower bone densities putting them at greater risk for osteoporosis in the future (Evans, Marel,

Lancaster, Kos, Evans, Wong, 1988; Ulrich, Georgiou, Snow-Harter, & Gillis, 1996; Francois, Benmaler, Guaydier-Souquieres, Sabatier, & Marcelli, 1999; Diaz, O'Neill, & Silman, 1997). Several studies have been conducted on mother-daughter pairs to identify familial similarities in osteoporosis risk. A study by McKay and colleagues (1994) observed positive mother-daughter correlations ranging from 0.57 ($p<0.05$) for the proximal femur to 0.38 (NS) for the third lumbar vertebrae. Additionally, a study done by Krall & Dawson-Hughes (1993) observed estimates of heritability at peripheral sites such as the radium and os calcis. These studies indicate that women who have a family history of osteoporosis should be encouraged to have bone density scans and to participate in preventive behaviors.

Although women with a family history of osteoporosis should be advised to care for themselves and to practice osteoporosis prevention behaviors, women without a family history should not consider themselves safe. Measures of family resemblance often do not clearly differentiate the relative contributions of shared environmental factors and genetic factors (Tudor-Locke & McColl, 2000). Genetic interactions may be as complex as inadequate vitamin D receptors to as simple as children emulating their parents' cooking and exercise behaviors. Additionally, in trying to assess one's risk of osteoporosis, simply considering the maternal side of the family tree may not be adequate.

An offspring is a combination of genes from two parents and a combination of behaviors from two parents. As a result, a person may be susceptible to the development of osteoporosis independent of family history. Consequently, every woman should be aware of her familial history related to osteoporosis, but she should also assume personal responsibility in her prevention of developing this disease.

Ethnicity and Osteoporosis

The incidence of hip fracture and osteoporosis varies widely among ethnic groups. The highest rates have been reported among whites of northern European ancestry (Luckey, Wallenstein, Lapinski, & Meier, 1996). Research indicates that women of Asian descent are also more likely to develop osteoporosis yet are less likely to experience osteoporotic hip fracture (Hirota, Nara, Ohguri, Manago, Hirota, 1992; Mackelvie, McKay, Khan, & Crocker, 2001). Studies report differences in body size, diet, and physical activity between Caucasian and Asian girls (Mackelvie et al, 2001). Typically, Asian girls consume about 480mg of calcium per day and participate in far fewer weight bearing activities than Caucasian girls. However, Asian girls also consume more plant based foods and plant sources of protein which may serve as a protective factor (Mackelvie et al., 2001).

Other studies have substantiated the reduced risk of hip fracture among Asian women as compared to Western Caucasian women. Life- and work-style differences between these cultures (i.e. sleeping on hard floors vs. beds, sitting on hard floors vs. couches, walking vs. driving) may be responsible for part of this reduced risk (Fujita, 1994). This is further supported with the increase in hip fractures among native Japanese women with the progressive Westernization of the Japanese lifestyle (Fujita, 1994).

Although Asian women seem to have less risk for osteoporotic hip fracture, it may not be due to the adherence to osteoporosis prevention behaviors. Studies have shown that Asian women have similar osteoporosis risk factors as Caucasian women, such as low calcium intake and lack of physical activity (Lau, Suriwongpaisal, Lee, De, Festin, Saw, et al., 2001). Additional risk factors found to be influential in the development of osteoporosis among Asian women were the low intake of dairy product in childhood (due to lactose intolerance), frequency of dieting, and skipping meals; all similar to those of Caucasian women (Hirota et al., 1992). However, the lack of osteoporotic hip fractures may be due to a lower center of gravity due to the shorter posture shared by many Asian women (Tudor-Locke & McColl, 2000).

Compared to those of African American or Hispanic ethnicities, White (non-Hispanic) and

Asian women are at greater risk for osteoporosis (Pun, Chan, Chung, & Wong, 1990). However, the mechanism for this difference is not known. Some research indicates that African American and Hispanic women achieve a higher peak bone density and lose bone density after menopause at a slower rate (Luckey et al., 1996).

Although research indicates that certain ethnicities are at a greater risk for osteoporosis than others, adherence to known osteoporosis prevention behaviors should be practiced. Ethnicity is not merely one's skin color. Ethnicity involves traditions, practices, and environmental influences (Tudor-Locke & McColl, 2000). Overall, osteoporosis is a multifaceted disease in which ethnicity is just a small fraction of the puzzle.

Medical History and Osteoporosis

As research in the area of osteoporosis continues to expand, new factors for concern are revealed. Within one's medical history, various medications have been demonstrated as protective or risk factors. Additionally, medical conditions such as eating disorders have also been shown to be potential risk factors for osteoporosis development.

Medications and Their Effects on Osteoporosis

Corticosteroids/ Glucocorticoids. The association between osteoporosis and corticosteroids or glucocorticoids was made shortly after the first use of these drugs in humans in the 1950s (Eastell, 1995). Corticosteroid treatments are so strongly related to the development of osteoporosis that they negate other factors that may be protective (i.e. race) (Lucasey, 2001). Corticosteroids are used for a variety of conditions such as asthma, rheumatoid arthritis, lupus, and inflammatory bowel disease. Concern is high among health educators because the number of young people who report using steroid medications is increasing.

Glucocorticoids affect bone in many ways. They adversely affect bone formation, bone resorption, calcium entry into the body in the gut and calcium exit from the body in the renal tubule (Reid, 1997). Osteoblasts are a group of bone cells primarily affected by glucocorticoids. These compounds affect osteoblasts by decreasing their proliferation, matrix synthesis, and decreasing their life span. This effect is thought to be mediated in part by a reduction in the production of local growth factors such as insulin-like growth factor 1 (Manolagas & Weinstein, 1999). When osteoblasts activity is hindered or decreased, it stands to reason that bone density will be compromised and thereby increase one's risk of fracture.

Research has also revealed that steroid-treated patients experience a state of calcium malabsorption (Klein, Arnaud, & Gallagher, 1977; Nordin, Marshal, Francis, & Crilly, 1981). Additionally, these patients experience a state of hypercalciuria which has been reported to be double of that in non-steroid using controls (Reid & Ibberston, 1987). Various therapy methods have been proposed to address the calcium deficit and imbalance. Among these therapies is calcium supplementation of 2000mg per day. Unfortunately, doses as high as 2400mg per day have shown to have no protective effect against the damage done by corticosteroids (Eastell, 1995; Reid, 1997; Yosipovitch, Hoon, & Leok, 2001).

In addition to causing bone loss, glucocorticoids appear to lead to changes in the architectural integrity of bone (Daens, Peretx, de Maertelaer, Moris, & Bergmann, 1999). Since bone loss occurs rapidly in the first 6 to 12 months of steroid therapy with a 5% to 20% decrease in bone density, it has been estimated that between 30% and 50% of long-term corticosteroid users will experience fractures (Lukert & Raiax, 1990).

Despite all the information about glucocorticoid-induced osteoporosis, few patients are provided with information about side effects and few are counseled on how to help prevent secondary osteoporosis. A study in England showed that only 6% of patients receiving glucocorticoid therapy received calcium supplementation (Peat, Healy, Reid, & Ralston, 1995) while another study out of England showed that only 14% of patients received prophylactic medication

(Walsh, Wong, Pringle, & Tattersfield, 1996). A further study conducted by Buckley and colleagues (1999) found that 58% of postmenopausal women received osteoporosis preventive treatment while using corticosteroids while premenopausal women and men were less likely to be treated.

Some researchers have argued that concern about steroid use should not be applied universally to all corticosteroids. There has been a lot of debate about the risks associated with oral glucocorticoids versus inhaled corticosteroids. Most of the data that exists correlates secondary osteoporosis and corticosteroid use based on the oral mode of administration. However, the few studies that have reviewed the effects of inhaled corticosteroids have identified osteoporosis risks as well. A study done Marystone and colleagues (1995) showed that oral steroid users had significant reductions in bone density when compared to non-steroid users. However, inhaled steroid users in the study showed intermediate reductions in bone mass when compared to non-steroid users although these values were not significant. Another study performed by Wong and colleagues (2000) showed a negative relation between total cumulative dose of inhaled corticosteroid and bone mineral density in patients with asthma.

Regardless of the route of administration, women who are utilizing corticosteroids, for any number of medical conditions, should receive counseling about ways to prevent secondary osteoporosis. Several behaviors should be adhered to and they are consistent with recommendations for non-steroid users in the prevention of osteoporosis. Women should receive primary prevention at the onset of corticosteroid therapy. This should include dietary advice to increase calcium and vitamin D while reducing sodium and caffeine intake. Women should also be counseled to participate in weight-bearing activities and to limit cigarettes and alcohol consumption. Additionally, women should receive a baseline bone density scan and be prescribed medications that have demonstrated a bone-protective role in the use of corticosteroids such as bisphosphanates (Eastell, 1995).

Depot Medroxyprogesterone (DMPA)/(Depo-Provera). DMPA is a progestin-only contraceptive that contains no estrogen. In 1992, it was approved by the Federal Drug Administration (FDA) and since then has been utilized by many women who struggle with contraceptive adherence or have a difficult time remembering to use contraception (Kass-Wolfe, 2001). There are numerous side effects of DMPA including menstrual irregularities including amenorrhea, weight gain, headaches, bloating of the abdomen or breasts, mood changes, and reduced libido (Kaunitz, 1998).

Depot medroxyprogesterone acetate (DMPA) is an injectable progesterone contraceptive technique that is used by over 3.5 million women in over 90 countries of the world (Cundy, Evans, Roberts, Wattie, Ames, & Reid, 1991; Mark, 1994). It works by primarily inhibiting ovulation through the suppression of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels and causing a decrease in estrogen levels (Kass-Wolff, 2001; Mark, 1994). As a result, ovulation stops and in many women, with continued use, they become amenorrheic (Cundy, et al., 1991). Since the recognition of estrogen deficiency as a cause of bone demineralization is widely accepted, there is growing concern about the potential development of osteoporosis and fractures with this contraceptive technique (Mark, 1994).

Contraceptive methods that decrease bone density in a population already deficient in calcium are a rising concern in women's health (Kass-Wolff, 2001). Studies indicate that women who use Depo-Provera, for a year or longer, have lower bone densities when compared to women who do not use this medication (Bahamondes, Perrotti, Castro, Faundes, Petta, & Bedone, 1999). Other studies have supported this finding. A study conducted by Cundy and colleagues (1991) demonstrated that users of DMPA for 5 or more years had a significant reduction in bone density as compared to nonusers: lumber spine -7.5% and femoral neck -6.6%.

The loss of bone density occurs rapidly when using Depo-Provera (Cundy & Reid, 1997). In a study by Cromer and colleagues (1996), the researchers compared subjects who used oral contraceptives, Norplant, and DMPA to non-contraceptive users. At the end of one year, the researchers found that bone density significantly decreased by 1.5% in the DMPA users when compared to the controls. Additionally, at the end of two years a significant bone density decrease of 3.1% was observed (Cromer, Blair, Mahan, Zibners, & Naumovski, 1996).
Research indicates that the use of Depo-Provera should be approached with caution. Counseling about this contraceptive is vital for women to understand the potential detrimental side effects. Although the loss of bone mass when using DMPA is considered to be temporary (Cromer et al., 1996) and reversible after treatment is discontinued, it is important to realize that the window of bone mass accrual is small and should be maximized. As a result, if bone density is decreased for 2-3 years due to DMPA use, these women are potentially at risk for achieving a lower peak bone mass due to this setback and therefore are at a greater risk of osteoporosis and fracture.

Oral contraceptives. Many young women choose oral contraceptives for various reasons other than their primary purpose of contraception. Oral contraceptives may be chosen to reduce menstrual cramps, regulate menstrual cycles, or as partial treatment for endometriosis. Research related to the effect of oral contraceptives as a means of osteoporosis prevention is inconclusive.

Several research studies have been conducted that show positive relationships between oral contraceptive use and the prevention of bone loss. These studies have been conducted on women at various ages and at various life stages. One of the most referred to studies was conducted by Kleerekoper and colleagues (1991). This cross-sectional, retrospective study of over 2200 women found that women with a history of oral contraceptive use were significantly more likely to have high bone mineral density measurements than those who did not have a history of oral contraceptive use (Kleerekoper, Brienza, Schultz, & Johnson, 1991). Additionally, a significant increase in bone mineral density was found with greater than 10 years of oral contraceptive use. The limitations of this study was that is did not control for smoking or exercise (Kleerekoper et al., 1991).

A study conducted by Lindsay, Tohme, and Kanders (1986) compared the bone density at various stages of life in women who ever used oral contraceptives versus those who never used oral contraceptives (OCs). An increase in bone mass of about 1% per year was observed among premenopausal women who had used OCs versus nonusers while there was no difference observed between matched-controls in the postmenopausal group (Linsay et al., 1986).

While many research projects indicate that oral contraceptives have an independent positive effect on bone density (Cooper, Hannaford, Croft, & Kay, 1993; Recker et al., 1992; Van Winter & Bernard, 1998), other research indicates no effect on bone density. In a study by Mazess and Barden (1991) the effect of oral contraceptive (OC) use on bone density was evaluated in 300 women between the ages of 20 and 39. The authors controlled for calcium intake, exercise, and smoking. No association was found between OC use and bone density.

Additional studies have not found a positive association between OC use and bone density. A study by Collins and colleagues (1988), found no significant differences in the lumbar bone mineral content, central density, or bone mineral density measurements between OC users and non-users. These subjects were matched for age, weight, and height. Furthermore, this study did not find a significant difference between the groups and the duration of oral contraceptive use which was up to 84 months in duration (Collins, Thomas, Harding, Cook, Turner, & Collins, 1988).

The variation in the findings of these studies can be related to many factors including study design, mode of bone density measurement, oral contraception composition, and duration of use. Many of the studies that reported positive

associations between bone mineral density and oral contraception use reviewed women who used OCs when they contained 50µg or more of estrogen. Today's oral contraceptive is considered low-dose and contains between 20µg and 40µg of estrogen. As a result, the lower dose of estrogen may prevent OCs from having positive effects on bone mineral density. Although they may not assist by increasing bone mineral density, OCs may slow down bone loss suppressing bone resorption which is evident in lower urinary calcium excretions (Shargil, 1985).

Research regarding osteoporosis prevention and oral contraception use is controversial. Those who may improve their bone density with the use of OCs are women who are already hypoestrogenic, have irregular menstrual cycles, or women who have other conditions that interfere with their estrogen producing capabilities. Although some protection may be offered for these special populations, women should not fall under the misperception that they are completely protected from osteoporosis development due to their use of OCs. Protecting oneself from this multifaceted debilitating disease requires attention to multiple prevention strategies.

Eating Disorders and Their Effects on Osteoporosis

Disordered eating refers to the spectrum of abnormal and harmful eating patterns used in a misguided attempt to lose weight or maintain a lowered body weight (Beals, Brey, & Gonyou, 1999). Because lower bone mineral density is one potential physiological consequence of eating disorders, the risk for osteoporosis among women afflicted with these diseases is greater (Clark, 1997). Both anorexia nervosa and bulimia nervosa and their associated behaviors increase a woman's risk for the development of osteoporosis.

Anorexia nervosa is a chronic illness that affects 1% of adolescent females and is characterized by a fear of fatness, self-imposed semistarvation and weight loss. Additionally, the illness has a high morbidity and is eventually fatal in 10-15% of cases (Seeman, Szmukler, Formica, Tsalamandris, & Mestrovic, 1992). Common clinical features of anorexia nervosa are estrogen deficiency (accompanied by amenorrhea) and a significant reduction in body weight (Treasure & Serpell, 2001).

Chronic anorexia nervosa is known to lead to osteopenia and osteoporosis in adults (Rigotti, Neer, Skates, Herzog, & Nussbaum, 1991; Seeman et al., 1992). Estrogen status is likely to be a major cause of osteopenia and osteoporosis in patients with anorexia nervosa (Treasure & Serpell, 2001). The occurrence of estrogen deficiency during the first three decades of life can increase the risk of osteoporosis by preventing the attainment of peak bone density and by causing accelerated bone loss (Seeman et al., 1992).

Other factors that may be related to the development of osteoporosis in women with anorexia nervosa are nutritional intake and physical activity. Due to the intense fear of being fat, many anorexics strictly limit their caloric intake. To limit their caloric intake they consume foods that are low calorie which oftentimes excludes foods rich in calcium such as dairy products (Treasure and Serpell, 2001). This extreme dietary limitation often limits their intake of other necessary nutritional components for bone health such as protein and vitamin D.

Besides severely limiting dietary intake, anorexics participate in excessive physical activity (Seeman et al., 1992). Although weight-bearing exercise can help protect against osteoporosis, excessive exercising can be associated with leanness and amenorrhea. Studies have been conducted to evaluate the effect of amenorrhea in young women suffering from anorexia nervosa. A study by Davies, Hall, and Jacobs, (1990) showed that the mean bone density was 15% lower for women with amenorrhea than age-matched controls and was related to the duration of amenorrhea and the severity of estrogen deficiency. This loss in bone density is not regained upon recovery from anorexia nervosa. A study by Hartman and colleagues (2000) found that for women who had been clinically recovered from anorexia nervosa on average of 21 years, their bone mineral density did not fully return to normal.

This was especially true when reviewing the BMD of the femur (Hartman, Crisp, Rooney, Rackow, Atkinson, & Patel, 2000).

The risk of osteoporosis occurring in other eating disorders has also been investigated. Studies reviewing women formally diagnosed with bulimia nervosa and other nonspecified eating disorders have been found to have significantly lower bone mineral densities than expected when compared to control data (Anderson, Woodward, & Lafrance, 1995). These results are not completely surprising because between 50%-60% of patients with a current diagnosis of bulimia nervosa have a previous history of anorexia nervosa (Fairburn & Hope, 1988).

While amenorrhea is a diagnostic criterion for anorexia, menstrual irregularities occur in only about half of patients with bulimia (Seidenfeld & Rickert, 2001). Although menstrual irregularities may not be enough to increase risk of osteoporosis in bulimic patients, the compensatory behaviors of self-inducing vomiting, abuse of laxatives and diuretics, abuse of diet pill, and caloric restriction can all affect bone health. Not consuming adequate calcium and other nutritional components necessary for bone health may influence the density and strength of bones later in life. However, at this time, no studies have identified an increased risk of osteoporosis fractures in previous or current bulimia nervosa patients (Seidenfeld & Rickert, 2001).

Summary

Osteoporosis is a multifaceted disease. Multiple factors have been implicated in the development of osteoporosis including dietary factors, heredity, ethnicity, lifestyle factors, medication use, and eating disorders. Although some of these factors are not alterable, the majority of them can be changed. Through the dissemination of osteoporosis prevention materials and information (see PowerPoint 1), health educators and other health professionals will increase knowledge about osteoporosis, increase osteoporosis prevention behaviors, and reframe the attitudes of women and men about their risk for developing this debilitating disease.

References

Abelow, B. J., Holford, T. R., & Insogna, K. L. (1992). Cross-cultural association between dietary animal protein and hip fracture: a hypothesis. Calcified Tissue International, 50, 14-18.

Alekel, D. L., St. Germain, A. S., & Peterson, C. T. (2000). Isoflavone-rich soy protein isolate exerts significant bone-sparing in the lumbar spine of perimenopausal women. American Journal of Clinical Nutrition, 72, 844-852.

American College of Sports Medicine [ACSM]. (1995). ACSM position stand on osteoporosis and exercise. Medicine and Science in Sports and Exercise, 27, i-viii.

Anderson, A. E., Woodward, P. J., & LaFrance, N. (1995). Bone mineral density of eating disorder subgroups. International Journal of Eating Disorders, 18, 335-342.

Anderson, J. B., & Metz, J. A. (1993). Contributions of dietary calcium and physical activity to primary prevention of osteoporosis in females. Journal of the American College of Nutrition, 12, 378-383.

Anderson, J. J. (1999). Plant-based diets and bone health: Nutritional implications. American Journal of Clinical Nutrition, 70, 539S-542S.

Anderson, J. J., Ambrose, W. W., & Garner, S. C. (1995). Orally dosed Genistein from soy and prevention of canellous bone loss in two ovariectomized rat models. Journal of Nutrition, 125, 799S.

Arden, N. (1997). Risk factors for osteoporosis. In: N. K. Arden & T. Spector (Eds.), Osteoporosis illustrated (pp. 326-352). London: Current Medical Literature.

Arney, W. K., & Pinnock, C. B. (1993). The milk mucus belief: Sensations associated with the belief and characteristics of believers. Appetite, 20, 53-60.

Aufdemorte, T. B. (1991). Pathologic assessment of bone. Hospital Practice, 26 (Suppl 1), 18-22.

Bahamondes, L., Perrotti, M., Castro, S., Faundes, D., Petta, C., & Bedone, A. (1999). Forearm bone density in users of Depo-Provera as a contraceptive method. Fertility and Sterility, 71, 849-852.

Baran, D., Sorensen, A., Grimes, J., Lew, R., Karellas, A., Johnson, B., et al. (1989). Dietary modification with dairy product for preventing vertebral bone loss in premenopausal women: A three-year prospective study. Journal of Clinical Endocrinology and Metabolism, 70, 264-270.

Barefield, E. (1996). Osteoporosis-related hip fractures cost $13 billion to $18 billion yearly. Food Review, 66, 31-36.

Barone, J. J., & Grice, H. C. (1994). Seventh International Caffeine Workshop. Food Chemical Toxicology, 32, 65-77.

Bass, M., Turner, L., & Hunt, S. (2001). Counseling female athletes: Application of the stages of change model to avoid disordered eating, amenorrhea, and osteoporosis. Psychological Reports, 88, 1153-1160.

Bassey, E. J., & Ramsdale, S. J. (1994). Increase in femoral bone density in young women following high-impact exercise. Osteoporosis International, 4, 72-75.

Beals, K. A., Brey, R. A., & Gonyou, J. B. (1999). Understanding the Female Athlete Triad: Eating disorders, amenorrhea, and osteoporosis. Journal of School Health, 69, 337-340.

Blalock, S. J., DeVellis, R. F., Giorgino, K. B., DeVellis, B. M., Gold, D.T., Dooley, M. A., et al. (1996). Osteoporosis prevention in premenopausal women: Using a stage model approach to examine the predictors of behavior. Health Psychology, 15, 84-93.

Boughton, B. (1999). Osteoporosis. Retrieved February 10, 2001, from http://web2.infotrac.galegroup.com/itw/i...dyn=7!xrn_3_0_A54823395?sw_aep=univofak2

Brandii, M. L. (1992). Flavonoids: Biochemical effects and therapeutic applications. Bone Mineralization, 19(Suppl), S3-S14.

Buckley, L. M., Marquez, M., Feezor, R., Ruffin, D. M., & Benson, L. L. (1999). Prevention of corticosteroid-induced osteoporosis: Results of a patient survey. Arthritis and Rheumatism, 42, 1736-1739.

Carter, D. R., Fyrie, D. P., & Whalen, R. T. (1987). Travecular bone density and loading history: Regulation of connective tissue biology by mechanical energy. Journal of Biomechanics, 20, 785-794.

Chapman, K. M., Chan, B. S., & Clark, C. D. (1995). Factors influencing dietary calcium intake in women. Journal of the American College of Nutrition, 14, 336-340.

Chapuy, M. C., Arlot, M. E., Duboeuf, F., Brun, J., Crouzet, B., Arnaud, S., et al. (1992). Vitamin D3 and calcium to prevent hip fractures in elderly women. New England Journal of Medicine, 327, 1637-1642.

Clark, K. (1997). Disordered eating behaviors and bone-mineral density in women who misuse alcohol. Western Journal of Nursing Research, 19, 32-55.

Collins, C. L., Thomas, K. A., Harding, A. F., Cook, S. D., Turner, J. L., Collins, J. H. (1988). The effect of oral contraceptives on lumbar bone density in premenopausal women. Journal of the Louisiana State Medical Society, 140, 31-32 and 35-39.

Combs, G. F. (1998). The vitamins: Fundamental aspects in nutrition and health (2nd ed.). San Diego, CA: Academic Press.

Conlisk, A. J., & Galuska, D. A. (2000). Is caffeine associated with bone mineral density in young adult women? Preventive Medicine, 31, 562-568.

Cooper, C., Hannaford, P., Croft, P., & Kay, C. R. (1993). Oral contraceptive pill use and fractures in women: A prospective study. Bone, 14, 41-45.

Cornuz, J., Feskanich, D., Willett, W. C., & Colditz, G. A. (1999). Smoking, smoking cessation, and risk of hip fracture in women. American Journal of Medicine, 106, 311-314.

Cromer, B. A., Blair, J. M., Mahan, J. D., Zibners, L., & Naumovski, Z. (1996). A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. Journal of Pediatrics, 129, 671-767.

Cromer, B. & Harel, Z. (2000). Adolescents: At increased risk for osteoporosis? Clinical Pediatrics, 39, 565-574.

Cummings, S. R., Rubin, S. M., & Black, D. (1990). The future of hip fractures in the United States: Numbers, costs, and potential effects of post-menopausal estrogen. Clinical Orthopedics, 252, 163-166.

Cundy, T., Evans, M., Roberts, H., Wattie, D., Ames, R., & Reid, I. R. (1991). Bone density in women receiving depot medroxyprogesterone acetate for contraception. British Medical Journal, 303, 13-16.

Cundy, T., & Reid, I. R. (1997). Bone loss and depot medroxyprogesterone. American Journal of Obstetrics and Gynecology, 176, 1116-1117.

Daens, S., Peretz, A., de Maertelaer, V., Moris, M., & Bergmann, P. (1999). Efficiency of quantitative ultrasound measurements as compared with dual-energy x-ray absorptiometry in the assessment of corticosteroid-induced bone impairment. Osteoporosis International, 10, 278-283.

Davies, M. C., Hall, M. L., & Jacobs, H. S. (1990). Bone mineral loss in young women with amenorrhea. British Medical Journal, 301, 790-793.

Dawson-Hughes, B., Harris, S. S., Krall, E. A., Dallal, G. E., Falconer, G., & Green, C. L. (1995). Rates of bone loss in post-menopausal women randomly assigned to one of two dosages of vitamin D. American Journal of Clinical Nutrition, 61, 1140-1145.

Dawson-Hughes, B., Harris, S. S., Krall, E. A., & Dallal, G. E. (1997). Effect of calcium and vitamin D supplementation on bone density in men and women 65 years of age or older. New England Journal of Medicine, 337, 670-676.

De Cree, C., Vermeulen, A., & Ostyn, M. (1991). Are high-performance young women athletes doomed to become low-performance old wives? The Journal of Sports Medicine and Physical Fitness, 31, 108-114.

Dequeker, J., Nijs, J., Verstraeten, A., Geusens, P., & Gevers, G. (1987). Genetic determinants of bone mineral content at the spine and radius: A twin study. Bone, 8, 207-209.

Devine, A., Criddle, R. A., Dick, I. M., Kerr, D. A., & Prince, R. L. (1995). A longitudinal study of the effect of sodium and calcium intakes on regional bone density in postmenopausal women. The American Journal of Clinical Nutrition, 62, 740-745.

Diaz, M. N., O'Neill, T. W., & Silman, A. J. (1997). The influence of family history of hip fracture on the risk of vertebral deformity in men and women: The European vertebral osteoporosis study. Bone, 20, 145-149.

Donohue, M. (1999). Osteoporosis prevention begins in childhood. Family Practice News, 29, 38.

Dowd, R. (2001). Role of calcium, vitamin D, and other essential nutrients in the prevention and treatment of osteoporosis. Nursing Clinics of North America, 36, 417-431.

Drinkwater, B. L., Bruemner, B., & Chestnut, C. H. (1990). Menstrual history as a determinant of current bone density in young athletes. Journal of the American Medical Association, 263, 545-548.

Eastell, R. (1995). Management of corticosteroid-induced osteoporosis. Journal of Internal Medicine, 237, 439-447.

Ervin, R. B., Wright, J. D., & Kennedy-Stephenson, J. (1999). Use of dietary supplements in the United States, 1988-1994. Vital and Health Statistics, 244, 1-14.

Evans, R. A., Marel, G. M., Lancaster, E. K., Kos, S., Evans, M., & Wong, S.Y. (1988). Bone mass is low in relatives of osteoporotic patients. Annals of Internal Medicine, 109, 870-873.

Fabbri, G., Petraglia, F., Segre, A., Maietta-Latessa, A., Galassi, M. C., Cellini, M., et al. (1991). Reduced spinal bone density in young women with amenorrhea. European journal of obstetrics & gynecology and reproductive biology, 41, 117-122.

Fairburn, C., & Hope, R. (1988). Disorders of eating and weight. In R. Kendell & A. Zealley (Eds.), Companion to psychiatric studies, (pp. 588-604). Edinburgh: Churchill Livingstone.

Fleming, K. H., & Heimbach, J. T. (1994). Consumption of calcium in the US: Food sources and intake levels. Journal of Nutrition, 124, 1426S-1430S.

Francois, S., Benmaler, A., Guaydier-Souquieres, G., Sabatier, J. P., & Marcelli, C. (1999). Heritability of bone mineral density. Reviews of Rheumatology, 66, 146-151.

Fujita, T. (1994). Osteoporosis in Japan: Factors contributing to the low incidence of hip fracture. Advances in Nutritional Research, 9, 89-99.

Gennari, C. (2001). Calcium and vitamin D nutrition and bone disease of the elderly. Public Health Nutrition, 4(2B), 547-559.

Gerrior, S., Putnam, J., & Bente, L. (1998). Milk and milk products: Their importance in the American diet. Food Review, 68, 29-37.

Glaser, D. L., & Kaplan, F. S. (1997). Osteoporosis: Definition and clinical presentation. Spine, 22 (24S), 12S-16S.

Hart, L., & Dip, P. G. (1996). Risk of osteoporosis in young women. Community Nurse, 2, 45.

Hartman, D., Crisp, A., Rooney, B., Rackow, C., Atkinson, R., & Patel, S. (2000). Bone density of women who have recovered from anorexia nervosa. International Journal of Eating Disorders, 28, 107-112.

Heaney, R. P. (1992). Calcium in the prevention and treatment of osteoporosis. Journal of Internal Medicine, 231, 169-180.

Heaney, R. P. (1993). Protein intake and the calcium economy. Journal of the American Dietetic Association, 94, 739-743.

Heaney, R. P. (1998). Excess dietary protein may not adversely effect bone. Journal of Nutrition, 128, 1054-1057.

Heaney, R. P. (2000). Dietary protein and phosphorus do not affect calcium absorption. American Journal of Clinical Nutrition, 72, 758-761.

Heaney, R. P., Dowell, M. S., Rafferty, K., & Bierman, J. (2000). Bioavailability of the calcium in fortified soy imitation milk, with some observations on method. American Journal of Clinical Nutrition, 71, 1166-1169.

Hegsted, D. M. (1986). Calcium and osteoporosis. Journal of Nutrition, 116, 2316-2319.

Hernandez-Avila, M., Colditz, G. A., Stampfer, M. J., Rosner, B., Speizer, F. E., & Willett, W. C. (1991). Caffeine, moderate alcohol intake, and risk of fractures o the hip and forearm in middle-aged women. American Journal of Clinical Nutrition, 54, 157-163.

Hirota, T., Nara, M., Ohguri, M., Manago, E., & Hirota, K. (1992). Effect of diet and lifestyle on bone mass in Asian young women. American Journal of Clinical Nutrition, 55, 1168-1173.

Holbrook, T. L. & Barrett-Connor, E. (1991). Calcium intake: Covariates and confounders. American Journal of Clinical Nutrition, 53, 741-744.

Hollenbach, K. A., Barrett-Connor, E., Edelstein, S. L., & Holbrook, T. (1993). Cigarette smoking and bone mineral density in older men and women. American Journal of Public Health, 83, 1265-1270.

Hopper, J. L., & Seeman, E. (1994). The bone density of female twins discordant for tobacco use. The New England Journal of Medicine, 330, 387-392.

Horwath, C. C., Bovern, C. H., Campbell, A. J., Busby, W., & Scott, V. (1995). Factors influencing milk and milk product consumption in young and elderly women with low calcium intakes. Nutrition Reviews, 15, 1735-1745.

Hsieh, C., Novielli, K. D., Diamond, J. J., & Cheruva, D. (2001). Health beliefs and attitudes toward the prevention of osteoporosis in older women. Menopause, 8, 372-376.

Institute of Medicine (IOM). (1997). Dietary Reference Intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington, DC: National Academy Press, 1997.

Jackman, L. A., Millane, S. S., Martin, B.R., Wood, O. B., McCabe, G. P., Peacock, M., et al. (1997). Calcium retention in relation to calcium intake and postmenarcheal age in adolescent females. American Journal of Clinical Nutrition, 66, 327-333.

Jamal, S. A., Ridout, R., Chase, C., Fielding, L., Rubin, L. A., & Hawker, G. A. (1999). Bone mineral density testing and osteoporosis education improve lifestyle behaviors in premenopausal women: A prospective study. Journal of Bone and Mineral Research, 14, 2143-2149.

Johansson, C., Mellstrom, D., Lerner, U., & Osterberg, T. (1992). Coffee drinking is a minor risk factor for bone loss and fractures. Age and Aging, 21, 20-26.

Johnston, C. C., Miller, J. Z., Slemenda, C.W., Reister, T. K., Hui, S., Christian, J. C., et al. (1992). Calcium supplementation and increases in bone mineral density in children. New England Journal of Medicine, 327, 82-87.

Kanders, B., Dempster, D. Q., & Lindsay, R. (1988). Interaction of calcium nutrition and physical activity on bone mass in young women. Journal of Bone and Mineral Research, 3, 145-149.

Kasper, M. J., Peterson, M. G., Allegrante, J. P., Galsworthy, T. D., & Gutin, B. (1994). Knowledge, beliefs, and behaviors among college women concerning the prevention of osteoporosis. Archives of Family Medicine, 3, 696-702.

Kass-Wolff, J. H. (2001). Bone loss in adolescents using Depo-Provera. Journal of the Society of Pediatric Nurses, 6, 21-31.

Katz, W. A., Sherman, C., & DiNubile, N. A. (1998). Osteoporosis. The Physician and Sportsmedicine, 26,33-36.

Kaunitz, S. M. (1998). Injectable depot medroxyprogesterone acetate contraception: An update for US clinicians. International Journal of Fertility, 43(2), 73-83.

Kiel, D. P., Felson, D. T., Hannan, M. T., Anderson, J. J., & Wilson, P. W. F. (1990). Caffeine and the risk of hip fracture. The Framingham Study. American Journal of Epidemiology, 132, 675-684.

Kleerekoper, M., Brienza, R. S., Schultz, L. R., & Johnson, C. C. (1991). Oral contraceptive use may protect against low bone mass: Henry Ford Hospital Osteoporosis Cooperative Research Group. Archives of Internal Medicine, 151, 1971-1975.

Klein, R. G., Arnaud, S. B., & Gallagher, J. C. (1977). Intestinal calcium absorption in exogenous hypercortisonism. Journal of Clinical Investigation, 60, 253-259.

Knapen, M. H. J., Hamulyak, J., & Vermeer, C. (1989). The effect of vitamin K supplementation on circulating osteocalcin and urinary calcium excretion. Annals of Internal Medicine, 111, 1001-1005.

Krall, E. A., & Dawson-Hughes, B. (1993). Heritable and lifestyle determinants of bone mineral density. Journal of Bone and Mineral Research, 8, 1-9.

Krall, E. A., & Dawson-Hughes, B. (1999). Osteoporosis. In M. E. Shils, J. A. Olson, M. Shike, & A. C. Ross (Eds.), Modern nutrition in health and disease (pp. 1353-1364). Baltimore, MD: Williams & Wilkins.

Krieger, N. S., Sessler, N. E., & Bushinsky, C. A. (1992). Acidosis inhibits osteoblastic and stimulates osteoclastic activity in vitro. American Journal of Physiology, 262, F442-F448.

Kulak, C. A., Schussheim, D. H., McMahon, D. J., Kurland, E., Silverberg, S. J., Siris, E. S., et al. (2000). Osteoporosis and low bone mass in premenopausal and perimenopausal women. Endocrine Practice, 6, 296-304.

Laitinen, K., Karkkainen, M., Lalla, M., Lamber-Allardt, C., Tunninen, R., Tahtela, R., et al. (1993). Is alcohol an osteoporosis-inducing agent for young and middle-aged women? Metabolism, 42, 875-881.

Laitinen, K., & Välimäki, M. (1993). Bone and the 'comforts of life'. Annals of Medicine, 25, 413-425.

Lappe, J. M. (1994). Bone fragility: Assessment of risk and strategies for prevention. Journal of obstetric, gynecologic, and neonatal nursing (JOGNN), 23, 260-268.

Lau, E. M. C., Suriwongpaisal, P., Lee, J. K., De, S. D., Festin, M. R., Saw, S. M., et al. (2001). Risk factors for hip fracture in Asian men and women: The Asian osteoporosis study. Journal of Bone and Mineral Research, 16, 572-580.

Lau, E. M. C. & Woo, J. (1998). Nutrition and osteoporosis. Current Opinion in Rheumatology, 10, 368-372.

Levin, R. J. (1999). Carbohydrates. In M. E. Shils, J. A. Olson, M. Shike, & A. C. Ross (Eds.), Modern nutrition in health and disease (pp. 49-65). Baltimore, MD: Williams & Wilkins.

Lewis, N. M., & Hollingworth, M. (1992). Food choice of young college women consuming low- or moderate-calcium diets. Nutrition Research, 112, 943-948.

Licata, A. A. (1992). Stress fractures in young athletic women: case reports of unsuspected cortisol-induced osteoporosis. Medicine and Science in Sports and Exercise, 24, 955-957.

Lindsay, R., & Dempster, D.W. (1985). Osteoporosis: Current concepts. Bulletin of the New York Academy of Medicine, 61, 307.

Lindsay, R., Tohme, J., & Kanders, B. (1986). The effect of oral contraceptive use on vertebral bone mass in pre- and post-menopausal women. Contraception, 34, 333-336.

Lloyd, T., Andon, M. B., Rollings, N., Martel, J., Landis, R., Demers, L. M., et al. (1993). Calcium supplementation and bone mineral density in adolescent girls. Journal of the American Medical Association, 270, 841-844.

Lloyd, T. Rollings, N., Eggli, D. F., Kieselhorst, K., & Chinchilli, V. M. (1997). Dietary caffeine intake and bone status of postmenopausal women. American Journal of Clinical Nutrition, 65, 1826-1830.

Lucasey, B. (2001). Corticosteroid-induced osteoporosis. Nursing Clinics of North America, 36, 455-466.

Luckey, M. M., Wallenstein, S., Lapinski, R., & Meier, D. E. (1996). A prospective study of bone loss in African-American and White women – A clinical research center study. Journal of Clinical Endocrinology and Metabolism, 81, 2948-2956.

Lukert, B. P., & Raiax, L. G. (1990). Glucocorticoid-induced osteoporosis: Pathogenesis and management. Annals of Internal Medicine, 112, 352-364.

Mackelvie, K. J., McKay, H. A., Khan, K. M., & Crocker, P. R. (2001). Lifestyle risk factors for osteoporosis in Asian and Caucasian girls. Medicine & Science in Sports & Exercise, 33, 1818-1824.

Manolagas, S. C. & Weinstein, R. S. (1999). New developments in the pathogenesis and treatment of steroid-induced osteoporosis. Journal of Bone and Mineral Research, 14, 1061-1066.

Marcus, R. (1982). The relationship of dietary calcium to the maintenance of skeletal integrity in man: an interface of endocrinology and nutrition. Metabolism, 31, 93-102.

Mark, S. (1994). Premenopausal bone loss and depot medroxyprogesterone acetate administration. International Journal of Gynecology and Obstetrics, 47, 269-272.

Mark, S., & Link, H. (1999). Reducing osteoporosis: prevention during childhood and adolescence. Bulletin of the World Health Organization, 77, 423-425.

Marystone, J. F., Barrett-Connor, E. L., & Morton, D. J. (1995). Inhaled and oral corticosteroids: Their effects on bone mineral density in older adults. American Journal of Public Health, 85, 1693-1695.

Masi, L. & Bilezikian, J. P. (1997). Osteoporosis: New hope for the future. International Journal of Fertility, 42, 245-254.

Massey, L. K. & Whiting, S. J. (1996). Dietary salt, urinary calcium and bone loss. Journal of Bone and Mineral Research, 11, 731-736.

Matkovic, V., Ilich, J. Z., Andon, M. B., Hsieh, L. C., Tzagournis, M. A., Lagger, B. J., et al. (1995). Urinary calcium, sodium, and bone mass of young females. American Journal of Clinical Nutrition, 62, 417-425.

Mazess, R. B., & Barden, H. S. (1991). Bone density in premenopausal women: Effects of age, dietary intake, physical activity, smoking, and birth-control pills. American Journal of Clinical Nutrition, 53, 132-142.

McCullock, R. G., Bailey, D. A., Houston, S., & Dodd, B. L. (1990). Effects of physical activity, dietary calcium intake and selected lifestyle factors on bone density in young women. Canadian Medical Association Journal, 142, 221-227.

McKay, H. A., Bailey, D. A., Wildinson, A. A., & Houston, C. S. (1994). Familial comparison of bone mineral density at he proximal femur and lumbar spine. Bone Minerals, 24, 95-107.

Mello, N. K., Mendelson, J. H., & Teoh, S. K. (1989). Neuroendocrine consequences of alcohol abuse in women. Annals of the New York Academy of Science, 562, 211-240.

Messina, M. (1995). Modern applications for an ancient bean: Soybeans and the prevention and treatment of chronic disease. Journal of Nutrition, 125, 567S-569S.

Messina, M. (1999). Legumes and soybeans: Overview of their nutritional profiles and health effects. American Journal of Clinical Nutrition, 70, 439S-450S.

Messina, M., Gardner, C., & Barnes, S. (2002). Gaining insight into the health effects of soy but a long way still to go: Commentary on the fourth International Symposium on the role of soy in preventing and treating chronic disease. Journal of Nutrition, 132, 547S-551S.

Micromedex. (2001). Calcium supplements. USP DI – Volume II, Advice for the patient, Annual 2001.

Mitchell, T. L., Wei, M., Gibbons, L. W., & Blair, S. N. (1999). Cardiorespiratory fitness and incidence of osteoporosis in women. Medicine and Science in Sports and Exercise, 31 (Suppl), S394.

Moon, M. A. (2000). Be aware that osteoporosis also affects young women and men. Family Practice News, 30, 25-26.

National Academy Press (NAP). (2000). Dietary Reference Intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Retrieved October 10, 2001, from http://www.nap.edu/openbook/0309063507/html/71.html

National Institutes of Health (NIH) Consensus Statement. (2000). Osteoporosis prevention, diagnosis, and therapy. Retrieved March 5, 2001, from http://odp.od.nih.gov/consensus/cons/111/111_statement.htm

National Osteoporosis Foundation (NOF). (1998). Strategies for osteoporosis. The Osteoporosis Report, Summer. Washington, DC: Author

National Osteoporosis Foundation (NOF). (2000). Boning up on osteoporosis: A guide to prevention and treatment [Brochure]. Washington, DC: Author

National Osteoporosis Foundation (NOF). (n.d.). Prevention: Calcium and vitamin D. Retrieved February 25, 2001, from http://www.nof.org/prevention/calcium.htm

Nordin, B. E. C., Marshal, D. H., Francis, R. M., & Crilly, R. G. (1981). The effects of sex steroid and corticosteroid hormones on bone. Journal of Steroid Biochemistry, 15, 171-174.

Notelovitz, M. (1993). Osteoporosis: Screening, prevention, and management. Fertility and Sterility, 59, 707-725.

Ooms, M. E., Roos, J. C., Bezemer, P., Van Der Vijgh, W. J., Bouter, L. M., Lips, P. (1995). Prevention of bone loss to vitamin D supplementation in elderly women: a randomized double-blind trial. Journal of Clinical Endocrinology and Metabolism, 80, 1052-1058.

Packard, P. T., & Recker, R. R. (1996). Caffeine does not affect the rate of gain in spine bone in young women. Osteoporosis International, 6, 149-152.

Pak, C. Y., Sakhaee, K., Adams-Huet, B., Piziak, V., Peterson, R. D., & Poindexter, J. R. (1995). Treatment of postmenopausal osteoporosis with slow-release sodium fluoride. Annals of Internal Medicine, 123, 401-408.

Pak, C. Y., Sakhaee, K., Rubin, C. D., & Zerwekh, J. E. (1997). Sustained-release sodium fluoride in the management of established postmenopausal osteoporosis. American Journal of Medical Sciences, 313, 23-32.

PDR Family Guide to Nutrition and Health. (1999). Sure fire ways to prevent brittle bones. Retrieved February 6, 2001, from http:web2infotrac.galegroup.com/itw/i…yn=10!xrn_4_0_A59040934?sw_aep+univofac2

Peat, I. D., Healy, S., Reid, D. M., & Ralston, S. H. (1995). Steroid induced osteoporosis: An opportunity for prevention? Annals of Rheumatic Diseases, 54, 66-68.

Pennington, J. A., & Schoen, S. A. (1996). Total diet study: Estimated dietary intakes of nutritional elements, 1982-1991. International Journal of Vitamin and Nutrition Research, 66, 350-362.

Peterson, J. A. (2001). Osteoporosis overview. Geriatric Nursing, 22, 17-22.

Plantalech, L., Guillaumont, M., Vergnaud, P., Leclercq, M., & Delmas P. D. (1991). Impairment of gamma carboxylation of circulating osteocalcin in elderly women. Journal of Bone and Mineral Research, 6, 1211-1216.

Pocock, N. A., Eisman, J. A., Hopper, J. L., Yeates, M. G., Sambrook, P. N., & Eberl, S. (1987). Genetic determinants of bone mass in adults: A twin study. Journal of Clinical Investigation, 80, 706-710.

Portsmouth, K., Henderson, K., Graham, N., Price, R., Cole, J., & Allen, J. (1994). Dietary calcium intake in 18-year-old women: Comparison with recommended daily intake and dietary energy intake. Journal of Advanced Nursing, 20, 1073-1078.

Powers, M. L., Heaney, R. P., Kalkwarf, H. J., Pitkin, R. M., Repke, J. T., Tsang, R. C., et al. (1999). The role of calcium in health and disease. American Journal of Obstetrics and Gynecology, 181, 1560-1569.

Pun, K. K., Chan, L. W. L., Chung, V., & Wong, F. H. W. (1990). Calcium and other dietary constituents in Hong Kong Chinese in relation to age and osteoporosis. Journal of Applied Nutrition, 42, 12-17.

Recker, R. R., Davies, M., Hinders, S. M., Heaney, R. P., Stegman, M. R., & Kimmel, D. B. (1992). Bone gain in young adult women. Journal of the American Medical Association, 268, 2403-2408.

Reid, I. R. (1997). Glucocorticoid osteoporosis – mechanisms and management. European Journal of Endocrinology, 137, 209-217.

Reid, I. R., & Ibberston, H. K. (1987). Evidence for decreased tubular reabsorption of calcium in glucocorticoid-treated asthmatics. Hormone Research, 27, 200-204.

Rico, H., Revilla, M., Villa, L. F., de Buergo, A.., & Arribas, I. (1994). Longitudinal study of the effect of calcium picolate on bone mass in eugonadal women. Calcified Tissue International, 54, 477-480.

Risco, F., Traba, M. L., & de la Piedra, C. (1995). Possible alterations of the in vivo 1,25(OH)2D3 synthesis and its tissue distribution in magnesium-deficient rats. Magnesium research, 8, 27-35.

Rigotti, N. A., Neer, R. M., Skates, S. J., Herzog, D. B., & Nussbaum, S. R. (1991). The clinical course of osteoporosis in anorexia nervosa: A longitudinal study of cortical bone mass. Journal of the American medical Association, 265, 1113-1138.

Robbins, S. P. & New, S. A. (1997). Markers of bone turnover in relation to bone health. Proceeding of the Nutritional Society, 56, 903-914.

Ross, P. D., Norimatsu, H., Davis, J. W., Yano, K., Wasnich, R. D., Fujiwara, S., et al. (1991). A comparison of hip fracture incidence among native Japanese, Japanese Americans, and American Caucasians. American Journal of Epidemiology, 133, 801-809.

Rubin, C. T., & Lanyon, L. E. (1985). Regulation of bone mass by mechanical strain magnitude. Calcified Tissue International, 37, 411-417.

Sabatini, S. (2001). The female athlete triad. American Journal of the Medical Sciences, 322, 193-195.

Schwartz, R., & Reddi, A. (1979). Influence of magnesium depletion on matrix-induced endochondrial bone formation. Calcified Tissue International, 29, 15-20.

Seeman, E., Szmukler, G. I., Formica, C., Tsalamandris, C., & Mestrovic, R. (1992). Osteoporosis in Anorexia Nervosa: The influence of peak bone density, bone loss, oral contraceptive use, and exercise. Journal of Bone and Mineral Research, 7, 1467-1474.

Seidenfeld, M. E. K. & Rickert, V. I. (2001). Impact of anorexia, bulimia and obesity on the gynecologic health of adolescents. American Family Physician, 64, 445-450.

Setchell, K. D. R. (2000). Absorption and metabolism of soy isoflavones – from food to dietary supplements and adults to infants. Journal of Nutrition, 130 (suppl 3), 654-655.

Shargil, A. A. (1985). Hormone replacement therapy in perimenopausal women with a triphasic contraceptive compound: A three year prospective study. International Journal of Fertility, 30, 15-28.

Siddiqui, N. A., Shetty, K. R., & Duthie, E. H., Jr. (1999). Osteoporosis in older men: discovering when and how to treat it. Geriatrics, 54, 20-25.

Slemenda, C. W., Christian, J. C., Reed, T., Reister, T. K., Williams, C. J., & Johnston, C. C. (1992). Long term bone loss in man effects of genetic and environmental factors. Annals of Internal Medicine, 117, 286-291.

Slemenda, C. W., Hui, S. L., Longcope, C., Wellman, H., & Johnston, C. C. (1990). Predictors of bone mass in perimenopausal women: a prospective study of clinical data using photon absorptiometry. Annals of Internal Medicine, 112, 96-101.

Smith, E., Gilligan, C., Smith, P., & Surpos, C. (1989). Calcium supplementation and bone loss in middle aged women. American Journal of Clinical Nutrition, 50, 833-842.

Smith, R. (1993). Bone physiology and the osteoporotic process. Respiratory Medicine, 87 (Suppl. A), 3-7.

Snow-Harter, C., Bouxsein, M. L., Lewis, B. T., Carter, D. R., & Marcus, R. (1992). Effects of resistance and endurance exercise on bone mineral status of young women: A randomized exercise intervention trial. Journal of Bone and Mineral Research, 7, 761-769.

Sowers, M. R., Wallace, R. B., & Lemke, J. H. (1986). The relationship of bone mass and fracture history to fluoride and calcium intake: A study of three communities. American Journal of Clinical Nutrition, 44, 889-898.

Sutton, N. A. (2000). The safety of calcium fortification. Medicine and Health, 83, 364-366.

Swaminathan, R. (1999). Nutritional factors in osteoporosis. International Journal of Clinical Practice, 53, 540-548.

Szulc, P., Arlot, M., Chapuy, M. C., Duboeuf, F., Meunier, P. J., & Delmas, P. D. (1994). Serum under-carboxylated osteocalcin correlates with hip bone mineral density in elderly women. Journal of Bone Mineral Research, 9, 1591-1595.

Szulc, P., Chapuy, M. C., Meunier, P. J., & Delmas, P. D. (1993). Serum under-carboxylated osteocalcin is a marker of the risk of hip fracture in elderly women. Journal of Clinical Investigation, 91, 1769-1774.

Taaffe, D. R., Robinson, T. L., Snow, C. M., & Marcus, R. (1997). High-impact exercise promotes bone gain in well-trained female athletes. Journal of Bone and Mineral Research, 12, 255-260.

Tavani, A., Negri, E., & LaVecchia, C. (1995). Coffee intake and risk of hip fracture in Northern Italy. Preventive Medicine, 24, 369-400.

Thomas, T. N. (1997). Lifestyle risk factors for osteoporosis. MedSurg Nursing, 6, 275-278.

Tokar, K., Ford, M. A., Turner, L. W., & Denny, G. (2003). Bone mineral density levels of college-aged women in Northwest Arkansas. Journal of the Arkansas Medical Society, 100, 170-175.

Toss, G. (1992). Effect of calcium intake vs. other life-style factors on bone mass. Journal of Internal Medicine, 231, 181-186.

Treasure, J., & Serpell, L. (2001). Osteoporosis in young people: Research and treatment in eating disorders. The Psychiatric Clinics of North America, 24, 359-370.

Tudor-Locke, C., & McColl, R. S. (2000). Factors related to variation in premenopausal bone mineral status: A health promotion approach. Osteoporosis International, 11, 1-24.

Turner, L. W., Leaver-Dunn, D., DiBrezzo, R., & Fort, I. (1998). Physical activity and osteoporotic fracture among older women. Journal of Athletic Training, 33, 207-210.

Turner, L. W., Taylor, J. E., & Hunt, S. (1998). Predictors for osteoporosis diagnosis among postmenopausal women: Results from a national survey. Journal of Women & Aging, 10(3), 79-96.

Turner, L. W., Bass, M. A., Ting, L., & Brown, B. (2002). Influence of yard work and weight training on bone mineral density among older U.S. women. Journal of Women and Ageing, 14, 139-148.

Ullom-Minnich, P. (1999). Prevention of osteoporosis and fractures. American Family Physician, 60, 194-203.

Ulrich, C. M., Georgiou, C. C., Snow-Harter, C. M., & Gillis, D. E. (1996). Bone mineral density in mother-daughter pairs: relations to lifetime exercise, lifetime milk consumption, and calcium supplements. American Journal of Clinical Nutrition, 63, 72-79.

Valimaki, M. J., Karkkainen, M., Lamber-Allardt, C., Laitinen, K., Alhava, E., Heikkinen, J., et al. (1994). Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass. Cardiovascular Risk in Young Finns Study Group. British Medical Journal, 309, 230-235.

Van Thiel, D. H. & Gavaler, J. S. (1990). Endocrine consequence of alcohol abuse. Alcohol, 25, 341-344.

Van Winter, J. T., & Bernard, M. F. (1998). Oral contraceptive use during the perimenopausal years. American Family Physician, 58, 1373-1377.

Vieth, R. (1999). Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety. American Journal of Clinical Nutrition, 69, 842-856.
Vuori, I. (1996). Peak bone mass and physical activity: A short review. Nutrition Reviews, 54, S11-S14.
Walsh, J. L., Wong, C. A., Pringle, M., & Tattersfield, A. E. (1996). Use of oral corticosteroids in the community and the prevention of secondary osteoporosis: A cross-sectional study. British Medical Journal, 313, 344-346.
Wardlaw, G. M. (1988). The effects of diet and lifestyle on bone mass in women. Journal of the American Dietetic Association, 88, 17-22.
Wardlaw, G. M. (1993). Putting osteoporosis in perspective. Journal of the American Dietetic Association, 93, 1000-1006.
Wardlaw, G. M. (1997). Contemporary nutrition: Issues and insights (3rd ed.). Iowa: Brown & Benchmark.
Wardlaw, G. M. & Weese, N. (1995). Putting calcium into perspective for your clients. Topics in Clinical Nutrition, 11, 23-35.
Weaver, C. M., & Heaney, R. P. (1999). Calcium. In M. E. Shils, J. A. Olson, M. Shike, & A. C. Ross (Eds.), Modern nutrition in health and disease (pp. 141-155). Baltimore, MD: Williams & Wilkins.
Welten, D. C., Kemper, H. C., Post, G. B., & Van Staveren, W. A. (1995). A meta-analysis of the effect of calcium intake on bone mass in young and middle aged females and males. Journal of Nutrition, 25, 2802-2813.
Whitney, E. N., & Rolfes, S. R. (2002). Understanding Nutrition (9th ed.). Belmont, CA: Wadsworth/Thomson Learning.
Willhite, L. (1998). Osteoporosis in women: Prevention and treatment. Journal of the American Pharmaceutical Association, 38, 614-623.
Wong, C. A., Walsh, L. J., Smith, C. J., Wisniewski, A. F., Lewis, S. A., Hubbard, R., et al. (2000). Inhaled corticosteroid use and bone-mineral density in patients with asthma. The Lancet, 355, 1399-1403.
Yosipovitch, F., Hoon, T. S., & Leok, G. C. (2001). Suggested rationale for prevention and treatment of glucocorticoid-induced bone loss in dermatologic patients. Archives of Dermatology, 137, 477-481.

Acknowledgements
I would like to thank Lori Turner, Mike Young, Ches Jones, Cathy Lirgg, and Kathleen Barta at the University of Arkansas for their continued support on this project. Your diligence and encouragement helped me to achieve my dream!

Author Information
Jeanne Freeman, PhD, CHES*
California State University, Chico
Department of Health and Community Services
Chico, CA 95929-0505
Ph. 530-898-5633
E-mail: jmfreeman@csuchico.edu

Lori Turner, PhD, RD
University of Arkansas
Department of Health, Kinesiology, Recreation & Dance
Fayetteville, AR 72701
Ph. 479-575-4670
E-mail: lori@uark.edu
* corresponding author